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# THE JOURNAL

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## The Maine Medical Association

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Pineland Hospital And Training Center Number

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a look  
at the  
literature



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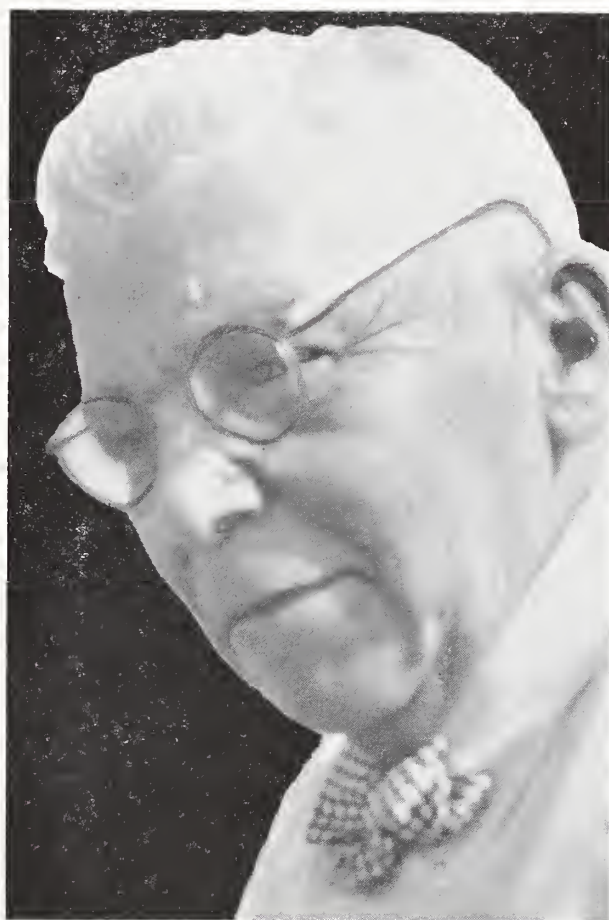
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Brunswick, Maine, January, 1962

No. 1

## Pineland Hospital And Training Center Number

*This edition is to honor Hans V. Mautner, M.D., of Pineland Hospital and Training Center, clinician, teacher, and scientist, on the occasion of his 75th birthday, May 9, 1961 — by his devoted friends and associates.*



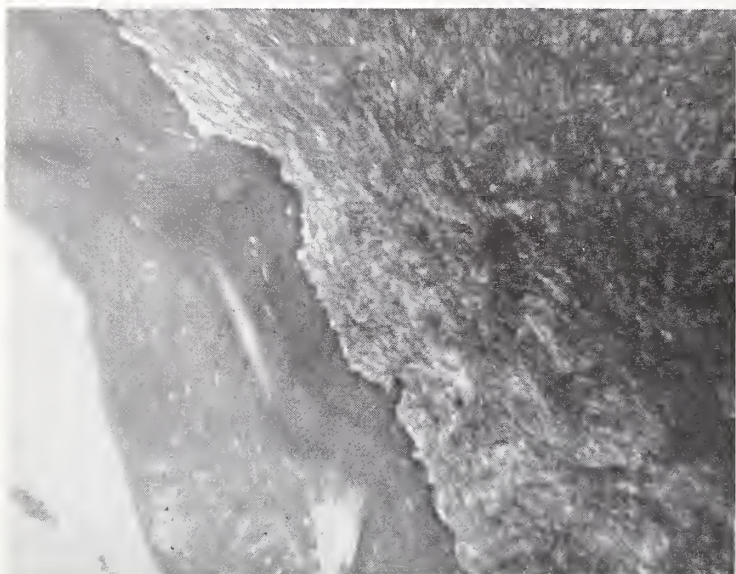


FIG. 5. Section through dura mater over left hemisphere showing bone trabecula with osteoblastic layer. Perdrau silver impregnation. x 100.

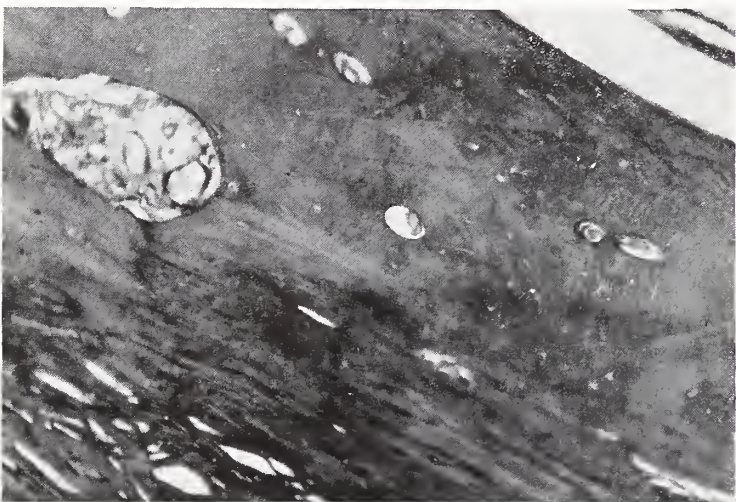


FIG. 6. Dura mater showing metaplastic bone formation. van Gieson-Elastica stain. x 150.

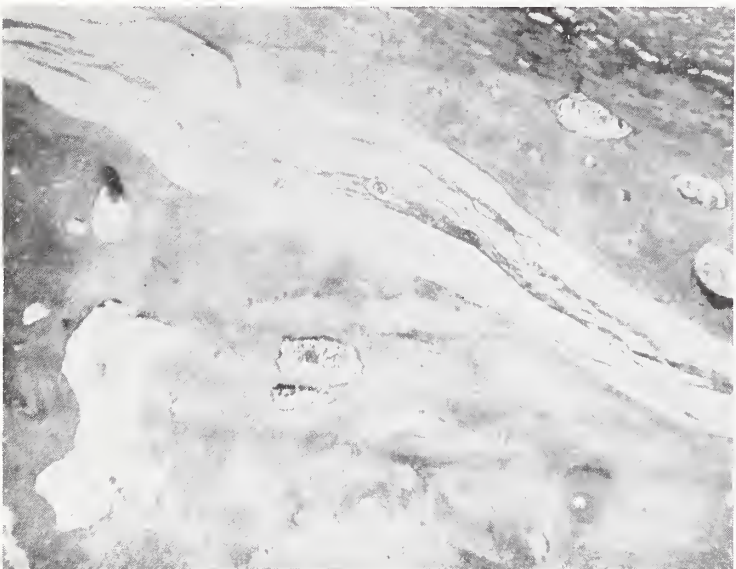


FIG. 7. Dura mater showing metaplastic bone formation (upper right). van Gieson-Elastich stain. x 44.

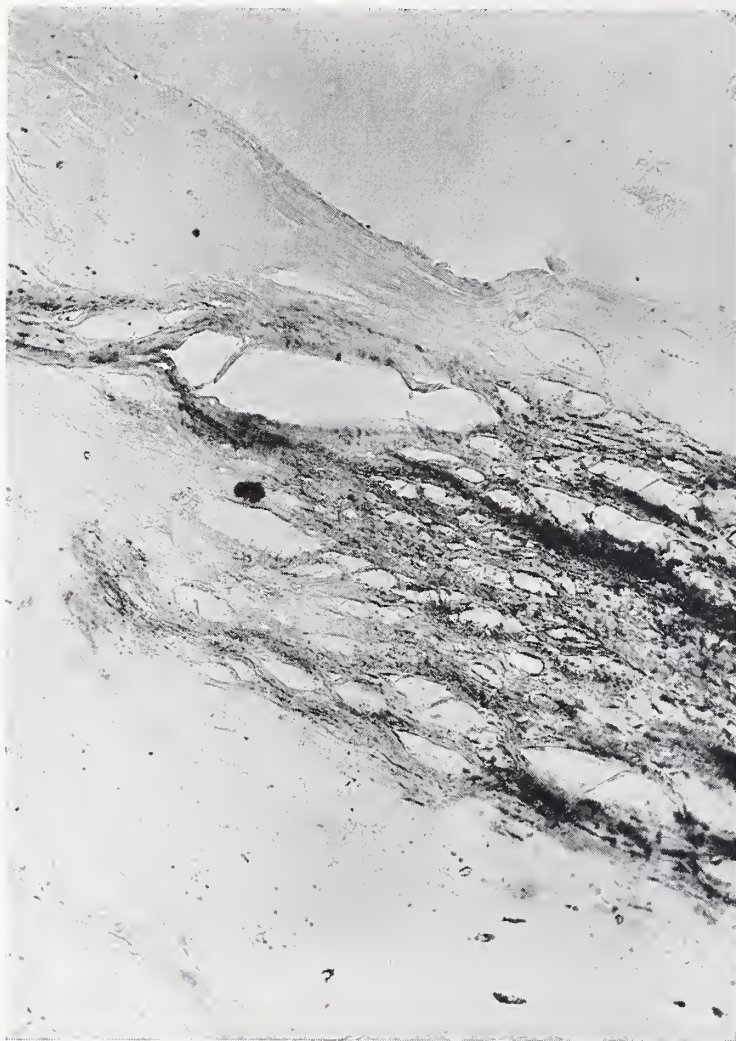


FIG. 8. Dura mater showing calcium incrustation of collagenous fibres as first change in metaplastic bone formation. Picric acid. x 160.

afterwards, the ground substance showed a change in staining properties: this appeared gradual in van Gieson-elastica stain, from dark purple through reddish to brown, while in Schmorl preparations the change appeared abrupt from blue to brown, though a slight bluish tinge could be noticed in the intermediate zone. In P. A. S. preparations the fibrous tissue undergoing metaplasia into bone tissue appeared deeper red than either the rest of the dura or the bone (Figs. 6, 7, 8, 9, 10, 11).

In some places the continuous layer of osteoblasts was interrupted by small area of direct metaplasia. In other places a bone trabecula showed direct metaplasia on one side and an osteoblastic bone formation on the other side.

The formation of filamentous processes by fibroblasts, thus their change into osteocytes was in some places seen to precede the change in the ground substance with appearance of osteocollagenous fibre-bundles and a ground substance with staining properties of bone. In such places cells with the morphology of osteocytes were seen in Schmorl-preparation to be situated between blue collagenous fibres. However, in most places of metaplastic bone formation, a calcium-incrustation of collagenous fibres appeared to be the initial change.



FIG. 9. Dura mater showing osteoblastic and metaplastic bone formation. H.E. x 160.

The brain showed two types of changes. The first, most marked in the fronto-parietal regio, showed a laminar and patchy (Fig. 12) loss of neurons associated in some places with a Chaslin gliosis. A severe neuronal loss was also seen in the cerebellum and involved in some places equally Purkinje cells and granular layer in other places mainly the Purkinje cells (Fig. 13). Demyelination and myelin sheath degeneration were most marked in the left fronto-parietal region (Fig. 14). The second type of change was the atrophy and distortion of basal ganglia on the left side, particularly severe in the head of the left caudate nucleus. This nucleus and also the lentiform nucleus and the thalamus of the left side were smaller than those of the right side and appeared displaced as if rotated outward. The internal capsule was on the left side about one quarter of that on the right side and partly demyelinated. In addition to the marginal gliosis of the cortex, there was also a subependymal gliosis in the left lateral ventricle. In the caudate nucleus and the thalamus bands of myelinated fibres were seen resembling a status mar-moratus. The spinal cord showed no abnormality.

#### DISCUSSION

Bone formation in the dura mater has already been described in two cases by *Morgagni* in his classical

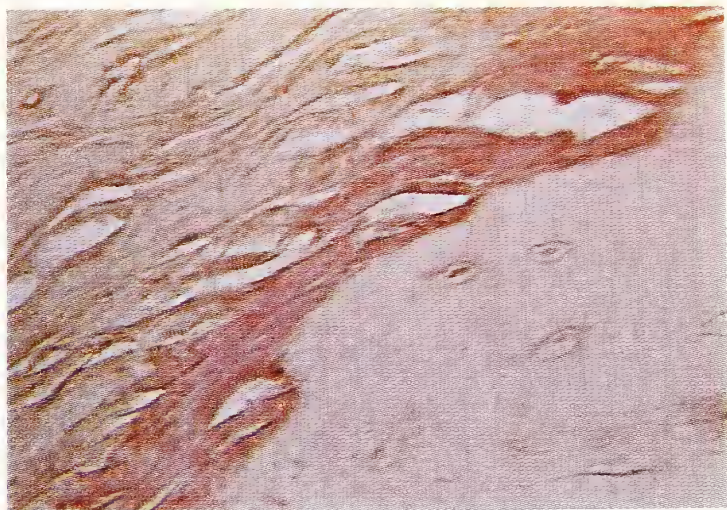


FIG. 10. Dura mater, showing change in staining properties of collagenous fibres. Neutral red. x 340.

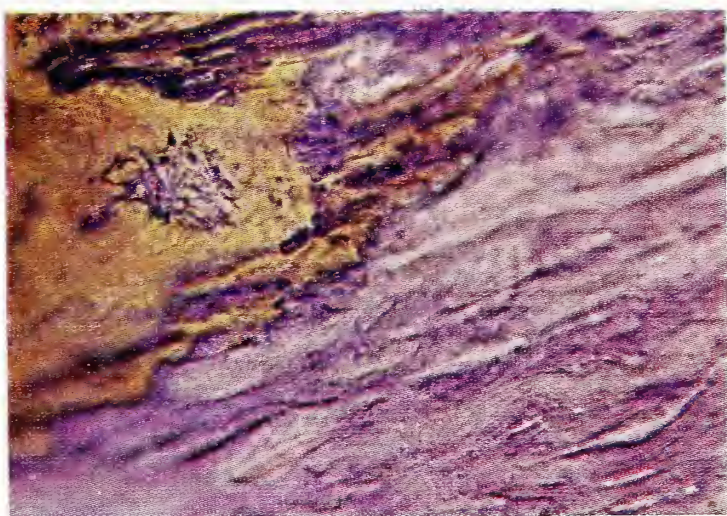


FIG. 11. Dura mater showing metaplastic bone formation. Schmorl's thionine-phosphomolybdic-picric acid. x 340.

book "de sedibus et causis morborum".<sup>1</sup> The largest number of bony plaques in dura mater was reported by *Jeannerat*<sup>2</sup> who found among 625 autopsies of mental patients ossifications of the dura in 64 cases. A complete review of the literature was subject of a Würzburg thesis by *Köster*<sup>3</sup> and more recently of a paper by *Halstead* and *Christopher*.<sup>4</sup> Most writers considered the bones in the dura mater as benign neoplasms, osteomata, or exostoses. As further possibilities were considered the origin in calcified thrombi and in "sand-bodies." The latter origin is suggested by the old age of the majority of patients. According to *Essbach*<sup>5</sup> the dura mater of people over 60 years of age shows almost constantly a deposition of fine fat droplets along the fibres which sometimes leads to the formation of dense concentric rings and occasionally to streaky calcification of fibres. Of the same origin are apparently the "fish-scale"-like multiple calcified plaques which are sometimes seen in the leptomeninges along the spinal cord of old people.

Conforming with the common teaching [*Maximow* and *Blum*,<sup>6</sup> *Weinmann* and *Sicher*<sup>7</sup>] that bone forma-



FIG. 12. Left precentral area, showing laminar neuronal loss. Holzer r. x 50.

tion is always due to resorption of preexisting tissue (calcified cartilage or fibrous tissue) followed by osteoid formation by specifically differentiated cells, the osteoblasts, it is generally assumed that the bone formation in the dura mater is also exclusively the result of osteoblastic activity. In the case presented in this paper areas are seen where typical Haversian bone is sharply demarcated against the fibrous tissue of the dura and lined by a continuous layer of flat cells, obviously inactive osteoblasts (Fig. 4). However, in other places it is clearly seen how fusiform fibroblasts become gradually transformed into osteocytes with slender processes and the ground substance gradually altered in its staining properties, becoming bone with lacunae and canaliculi (Fig. 11). In van Gieson-elastica preparations the purple and black colour of the fibro-elastic tissue is gradually replaced by the brown colour of bone. In Schmorl's stain the colour change appears abrupt though a slight bluish tinge can be seen in an intermediate zone. Thus, a transformation of fibrocytes into osteocytes in bone lacunae and of collagenous and elastic into bone-ground substance, without a layer of osteoblasts, has taken place, or a direct metaplasia. Nowhere remainders of elastic membranes of blood vessels were seen, nowhere vascular structures were demonstrable by the Perdrau stain, and only very occasionally a few granules of haemosiderin could be detected. In no place of the numerous examined sections of the dura

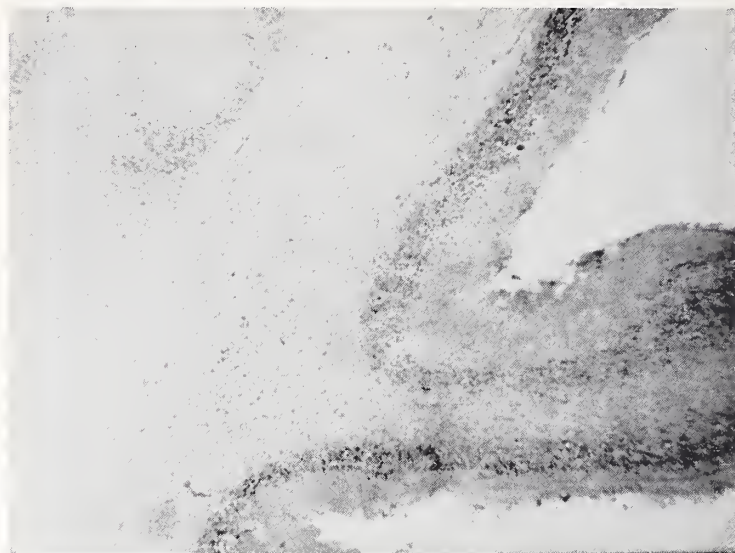


FIG. 13. Cerebellum showing loss of Purkinje cells and atrophy of granular layer. Nissl stain. x 44.



FIG. 14. White matter of left pericentral area showing degeneration of myelin sheaths. Luxol blue. x 44.

mater calcium deposits in the fibrous tissue were seen. There is, therefore, no evidence of origin in a haemangioma of dura mater with calcified thrombi. The localization of the bone is different from the distribution of blood vessels in the developing meninges as described by *Streeter*.<sup>8</sup> The findings exclude also the origin from a calcified dural haematoma. The multiplicity of bone formation in one localization (dura over the vertex), the presence of convulsions since early infancy, the absence of progression, the normal structure of Haversian bone, all militate against a neoplastic growth and favour the hypothesis of a developmental error, a tissue malformation, a hamartoma or hamartosis in the definition of *Albrecht*.<sup>9</sup> *Willis*<sup>10</sup> gives the same interpretation for the majority of haemangiomas. *Halstead* and *Christopher*<sup>1</sup> mention among the possibilities for bone formation in dura mater the "retention of the osteogenic function by islands of dura" and *Henschen*<sup>13</sup> discussed on a similar basis the bone formation in meningiomas.

Obviously, a developmental error need not always be the cause of duraossification. Thus, in the case of *Thi-*

*loix* and *DuPasquier*<sup>14</sup> an ossified plaque 8 x 6 inches in size was apparently due to congestion resulting from cerebral haemorrhage and demonstrates the latent osteogenic potentialities of dural fibrous tissue. Of different origin is also degenerative ossification of dura in old people which is the comparatively frequent form of dural ossification.

It need scarcely be emphasized that the explanations given for the bone formation in the dura mater of the presented case are valid only for the latter and not for the leptomeninges. The old theory of *His* and *Kölliker* that all meninges develop from a mesenchymal meninx primitiva is supported by the investigations of *Essbach* who claims that the meninges develop from a uniform cellular tissue which later, in the "skeleto-neural" intermediate stage, differentiates into a loose mesenchyma of the leptomeninx and a more dense mesenchyma of the pachymeninx in which a further condensation into the future periosteum can be observed. However, most of the more recent investigators (*Henschen*) accept the theory of *Oberling*, *Masson*, *Roussy* and associates that most cellular elements of the leptomeninx are of gliectodermal origin and only the blood vessels the mesenchymal component. The most convincing experiment is that of *Harvey* and *Burr* who observed development of pia mater only if the tissue transplanted to another animal included elements of the ganglionic crest. *Juba*, *Mayer* and *Krücke* accept this theory and claim that bone in the leptomeninges develops from thrombosed arteries in which ossification starts in the lamina elastica interna.

The severe changes in the brain, as described above, are in part identical with those generally seen in patients who suffered for a long time from epileptic fits. These changes have been studied by *Scholz* and by *Meyer* and named by the former "Krampfschädigungen." They include laminar and patchy loss of neurons, loss of Purkinje cells, atrophy of granular layer of cerebellum, neuronal loss in the Sommer sector of hippocampus and marginal gliosis. They appear to be the result of anoxia during the fits. It is worthy of note that these changes were most severe in the atrophic left hemisphere, particularly in the fronto-parietal area. Though postepileptic hemiatrophy of the brain is known to occur, in the present case it is probably the direct result of bone formation in the dura mater. The various explanations of the hemiatrophy of the brain — including that associated with a status spongiosus — are discussed by *Norman*. It appears that hemiatrophy of the brain has not a uniform aetiology and that the main causes are vascular changes. In the present case the hemiatrophy is on the same side as the most severe ossification of the dura. From the size and the distortion of the basal ganglia it may be concluded that interference was present in the stage of development. It is, therefore, suggested that the hemiatrophy was the result of the space occupying intracranial lesion interfering with growth and development. With the closure of sutures

the cranial cavity becomes inexpandable and the space occupying bones of the dura mater will cause a decrease in the volume of blood and/or cerebrospinal fluid. This decrease in nutritional fluids will result in retardation and arrest in the development of cerebral tissue.

There is no support for the assumption that the disease at the age of four months was a meningoencephalitis and the epilepsy its result.

#### SUMMARY

The case of a thirty-year old female is described who suffered from epileptic fits since early infancy and hemiplegia. The post-mortem examination revealed extensive bone formation in the dura mater and hemiatrophy of the brain. The bone formation was partly due to osteoblastic activity and partly to direct metaplasia. The brain showed changes characteristic for epilepsy and, in addition, atrophy of gyri, demyelination, atrophy and distortion of basal ganglia. The latter findings are explained as the result of decreased supply with nutritional fluids during the developmental stage.

I wish to thank Mrs. E. Beverage for technical assistance, Mrs. P. Mundy for clerical work, and Mr. G. Jacobs for photography.

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# Synopsis Of Some Current Studies In Phenylketonuria\*

HANNS-DIETER GRÜMER, M.D.\*\*

In 1934 a previously unknown disease was described by the Norwegian physiologist, Fölling.<sup>27</sup> He reported 10 cases, characterized by mental deficiency and the excretion of phenylpyruvic acid in the urine, and he called the disease "Imbecillitas phenylpyrouvica." Jervis later proposed the name phenylpyruvic oligophrenia, and, although this latter name has generally been accepted in the Anglo-American literature, the condition has sometimes been referred to as Fölling's disease in honor of its discoverer.

Twenty years after the discovery of phenylpyruvic oligophrenia, Jervis<sup>47</sup> listed 513 published cases. From his study he estimated the incidence of phenylketonuria among institutionalized retardates to be 0.65%. According to this author, about 40 (20-60) patients per million in the general population are afflicted. Since 1956 we have observed 20 phenylketonurics out of 1,907 retarded and institutionalized patients (1.05%), 15 of whom lived in our institution (0.79%). At the present time we have 18 phenylketonuric patients in our institution and 5 outpatients. Since the total population of Maine amounts to about one million, our figures suggest that the occurrence of phenylketonuria in the State is close to the lower limit given above. Blehová<sup>18</sup> recently tested 2,444 mentally defectives in institutions of Bohemia and Moravia and found 10 phenylketonuric individuals among them (0.41%).

## CLINICAL FINDINGS

For a better understanding of the metabolic abnormalities found in phenylketonuria, a short description of the clinical and pathological findings may be helpful.

Most of the untreated patients afflicted with this disease are of very low-grade intelligence. Rarely patients with borderline or average intelligence are seen, and only 15 such patients with measured intelligence quotient (I.Q.) were recently summarized by Knox<sup>54</sup> and Tischler et al.<sup>73</sup> About 63% have an I.Q. of below 21, 36% between 21 and 70, and only about 1% an I.Q. of above 70.<sup>47</sup> Jervis<sup>47</sup> noticed marked neurological changes in about one-third of his phenylketonuric cases, only slight deviations in one-third, and normal limits in the remaining one-third. These neurological changes included hyperactivity of the deep re-

flexes, muscular hypertonicity, awkward or rigid gait, tremors, clonus of foot or rotula. Pyramidal signs have not been found.

Phenylketonuric patients often are easily irritated by their surroundings, especially in a large ward and frequently respond to noise (with temper tantrums or destructive behavior, for instance). Epileptic seizures are frequently observed in infancy and in early childhood, but rarely in the adult phenylketonuric,<sup>47,51,57</sup> and minor electroencephalogram abnormalities are commonly seen in untreated cases. Upon entering a room where phenylketonuric patients are present, a characteristic mouse-like or musty odor is easily detected which may be attributed to the excretion of phenylacetic acid. The low-grade phenylketonurics, in particular, prefer to sit in a tailor-like position and rock back and forth, sometimes very vigorously. The hands and arms are also constantly in motion, and a grinding of the teeth may be heard. The majority have blue eyes and blond hair (or at least lighter than the non-afflicted family members) even when the parents are of dark complexion. The skin is frequently eczematous. In our own observations it was noted that the stature of these patients is slightly smaller than average; thus they appear to be younger than their chronological age. However, no gross abnormalities have commonly been found.

Values for phenylalanine and its derivative, phenylpyruvic acid, in blood, liquor cerebrospinalis, and urine have been reviewed by Jervis<sup>47</sup> and are summarized in Table I. A more detailed account of the values with various methods is given by Knox.<sup>54</sup>

Excretion of phenylpyruvic acid can easily be detected by the addition of a few drops of 5 to 10% ferric chloride solution to the acidified urine. A green, fading color indicates a positive result. This test becomes positive within the first 6 weeks of life.

In 1954, Jervis<sup>47</sup> listed a series of publications in which essentially negative findings of the central nervous system were reported in autopsies, and he assumed that the striking demyelination described by Benda<sup>9</sup> in 2 cases must be exceptional. However, more cases with marked demyelination have been described by Alvord and co-workers,<sup>2</sup> and by Poser and Van Bogaert,<sup>69</sup> who believe that in phenylketonurics the formation of myelin fibers is disturbed rather than that a destruction of myelin sheaths takes place. Four patients upon whom autopsies were performed at Pineland Hospital showed extensive areas of demyelination, but no pre-

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TABLE I

Concentration of phenylalanine and phenylpyruvic acid in blood, cerebrospinal fluid and urine of phenylketonuric patients. The numbers in brackets are normal values.

	<i>serum</i> (mg. per 100 ml.)	<i>liquor</i> (mg. per 100 ml.)	<i>urine</i> (mg. per 24 hrs.)
phenylalanine	10-35 (0.5-1.5) *	6-8	0.3-1 (0.16)
phenylpyruvic acid	0.2-1.4 (0)	0 (0)	0.3-2 (0)

\* In personal observations with the method by LaDu for phenylalanine in plasma we found values ranging from 24-65 mg per 100 ml with an average of 43.

disposition of certain areas could be established<sup>7</sup> as the localization of demyelination was different in each case.

For further clinical details, the reader should refer to the excellent reviews in the English literature by Jervis in 1954,<sup>47</sup> Mautner in 1959,<sup>60</sup> and Knox in 1960.<sup>54</sup> No essentially new clinical findings have since been added.

### BIOCHEMICAL FINDINGS

The story of phenylpyruvic oligophrenia is remarkable and one of the turning points in the history of mental deficiency. Fölling detected this disease merely by chance.\* He was interested in the investigation of ketone bodies in human urines. For the detection of the keto group he used, among other reagents, ferric chloride, which gave a green color in the acidified urine of two siblings suffering from mental deficiency. This color-forming reaction was not seen in controls. He and his associates identified the abnormal compound as phenylpyruvic acid and were later able to demonstrate that phenylalanine was present in unusually large amounts in the blood and urine of phenylketonuric patients.<sup>28</sup> They concluded that phenylpyruvic acid was formed from L-phenylalanine which, when administered orally, raised the concentration of phenylalanine in blood.<sup>29</sup> About a decade later, Jervis<sup>46</sup> was able to localize the position of the metabolic error in phenylketonuria. Ingestion of phenylalanine or of phenylpyruvic acid resulted in an increase of Millon positive compounds (tyrosine) in the blood of normals. In contrast to this, phenylketonuric patients did not demonstrate an increase of Millon substances. This finding could only be interpreted as a block of the conversion of phenylalanine to tyrosine (Fig. 1). Six years later, Udenfriend and Bessman<sup>74</sup> confirmed this observation. They fed C<sup>14</sup>-labeled phenylalanine to phenylketonuric and to normal individuals and isolated phenylalanine and tyrosine from plasma proteins. The molar ratio of the radio-activity of tyrosine to phenylalanine was about 0.02 in phenylketonurics and about 0.23 in normals.

As early as 1913, Embden and Bades<sup>25</sup> had demonstrated the conversion of phenylalanine to tyrosine in liver perfusion experiments. This was confirmed by experiments of Moss and Schoenheimer<sup>66</sup> when they

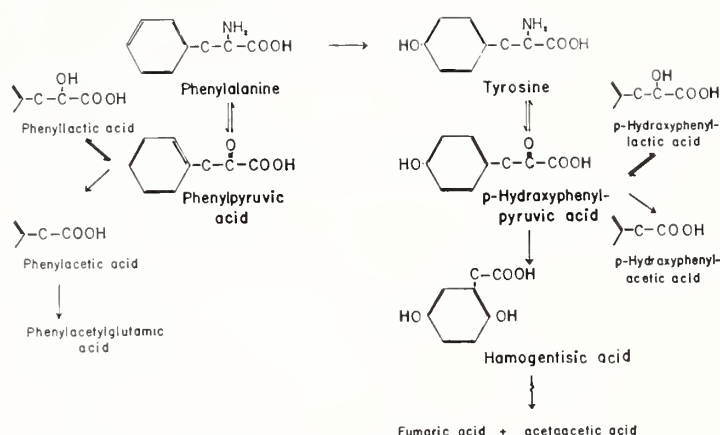


FIG. 1. Formation of homogentisic acid from phenylalanine and its derivatives.

isolated labeled tyrosine from proteins after feeding deuterium containing phenylalanine to growing and to adult rats. Udenfriend and Cooper<sup>75</sup> gave evidence of the high specificity of the enzyme system converting phenylalanine to tyrosine. Purification of this system revealed two enzymes; phenylalanine hydroxylase I which is labile and only found in the liver and the more stable, but non-specific, phenylalanine hydroxylase II which is also found in other tissues.<sup>63</sup> Wallace and co-workers,<sup>79</sup> as well as Mitoma and others,<sup>64</sup> reported the absence of phenylalanine hydroxylase I activity from liver of phenylketonurics. Studies of the phenylalanine hydroxylating co-factors are in progress.<sup>49,50,51,52,53</sup> Tetrahydropteridine, a folic acid derivative, and tetrahydrofolic acid can substitute for the natural co-factor and are kept in the reduced state by phenylalanine hydroxylase II. Folic acid antagonist, e.g., aminopterin and amethopterin, inhibit the conversion of phenylalanine to tyrosine in rats. Amethopterin has also an inhibitory effect on the phenylalanine conversion in humans,<sup>30,31</sup> suggesting that a pteridine co-factor is active in hydroxylation of phenylalanine in man. Newborn rats are not capable of converting phenylalanine to tyrosine, but develop this ability to the level of adults several days after birth.<sup>70</sup>

Studies of the effect of high concentrations of phenylalanine and its non-parahydroxylated derivatives on metabolic processes have been undertaken. It is hoped from these studies to obtain further information about the metabolic disorders characteristic of the phenylketonuric organism, and to find some lead to the prob-

\*Personal communication

lem of why phenylketonuric individuals are mentally deficient. It is surprising to find only a few publications regarding the influence of these substances on brain metabolism. Hanson<sup>35</sup> studied the effect of phenylalanine and its derivatives on brain glutamic acid decarboxylase. The tested inhibitors were made up to concentrations of about 135 mg per 100 ml final homogenate. A distinct inhibition of glutamic acid decarboxylase was found by some of these substances, but phenylalanine itself had no inhibitory effect, not even at the high concentrations used in their experiments. Himwich and Fazekas<sup>36</sup> observed a slightly diminished oxygen uptake by brain tissue in phenylketonuric patients *in vivo* as measured by the arterio-venous oxygen difference of carotid artery and jugular vein. Wortis<sup>83</sup> stated that he found a depressed oxidative activity of minced rat brain in the Warburg apparatus after the addition of phenylketonuric serum. Unfortunately, insufficient experimental data were presented to support the significance of his statement.

Bickis, Kennedy, and Quastel<sup>16</sup> examined the effect of phenylalanine on the breakdown of tyrosine to acetoacetate in rat liver slices. The concentrations of phenylalanine used for inhibition were 2 mM (= 33 mg per 100 ml final solution). Such concentrations correspond to those determined in the protein-free plasma fraction of phenylketonuric patients. A diminished formation of acetoacetate was found by these authors in their *in vitro* system. However, Grümer and Woodard<sup>34</sup> did not find a diminished breakdown of the tyrosine derivative p-hydroxyphenylpyruvic acid, when this substance was given orally to phenylketonuric individuals. Reduced formation of pigments from L-tyrosine by mushroom tyrosinase was seen by Dancis and Balis<sup>23</sup> in inhibition studies with L-phenylalanine when the molar ratio of phenylalanine to tyrosine was 10 to 1 or higher. Slight inhibition by 1 mM phenylalanine of the L-tyrosine to L-dihydroxyphenylalanine converting enzyme from the Harding-Passey melanoma has also been reported by Miyamoto and Fitzpatrick;<sup>65</sup> the d-isomer of phenylalanine has *no* significant inhibitory effect on tyrosinase.

In order to draw conclusions of biological significance from inhibition studies, the concentration of this inhibitor within the intact cell has to be ascertained. Grümer, Koblet, and Woodard<sup>33</sup> studied the distribution of C<sup>14</sup>-labeled phenylalanine between plasma and tissue of phenylketonuric subjects. Their results do not indicate a substantial intracellular enrichment of phenylalanine above plasma levels. In other words, the range of free phenylalanine concentrations, as they occur in the plasma of phenylketonuric patients, seems to be optimal for *in vitro* studies. These studies also provided information on the amount of the free phenylalanine pool, that is, the non-protein bound phenylalanine in normal and phenylketonuric subjects. It was found that a normal individual had a free phenylalanine pool of about 1 g, while a phenylketonuric subject of

70 kg body weight with a free phenylalanine concentration of 47 mg per 100 ml plasma had a free phenylalanine pool of 15.5 g. The experiments indicated a direct correlation between the free plasma phenylalanine concentration and the magnitude of the free phenylalanine pool. A rough estimate of the accumulated free phenylalanine within the body can be obtained if the free phenylalanine content of plasma and the water space are known; that is, the product of free phenylalanine in 1.0 ml plasma or serum and the water (antipyrine) space in ml.

Armstrong and Robinson<sup>4</sup> noted in most phenylketonuric patients an increased urinary excretion of indoleacetic acid and indolelactic acid, whereas, low levels of 5-hydroxytryptamin (serotonin) in serum and of 5-hydroxyindoleacetic acid in urine of this group of patients were reported by Pare et al.<sup>67</sup> Excretion of indolelactic acid and indoleacetic acid ceased when a phenylalanine restricted diet was given.<sup>81</sup> Phenylalanine has an inhibitory effect *in vitro* on 5-hydroxytryptophan decarboxylase<sup>24</sup> which converts 5-hydroxytryptophan to 5-hydroxytryptamin. Tolerance tests with 5-hydroxytryptophan revealed also *in vivo* inhibition of 5-hydroxytryptophan decarboxylase in phenylketonuric patients as compared to non-phenylketonuric, but otherwise retarded, controls.<sup>68</sup> When phenylketonuric individuals were placed on a diet low in phenylalanine, a normalization of the 5-hydroxytryptamin plasma concentration<sup>68</sup> and of the urinary excretion of 5-hydroxyindoleacetic acid<sup>8</sup> was seen. In preliminary testing Baldridge and co-workers<sup>8</sup> noticed a "spurt in mental development" in a 38-months-old patient to whom 100 mg of 5-hydroxytryptophan was administered daily along with a normal diet. However, no further information was given and it remains to be seen whether this observation can be confirmed in more cases. In this connection, it is of interest to note that treatment with 5-hydroxytryptophan strikingly improved the EEG pattern in patients with hepatic coma.<sup>19</sup> Based on the observations of Armstrong and Robinson, as well as Pare's group, Bessman and Tada<sup>11</sup> found that in phenylketonuria the excretion of indican paralleled the blood level of phenylalanine. The urinary indican-creatinine index was considered as the most reliable sign of increased indicanuria. If indole was given orally the net conversion of indole to indican was somewhat greater in the control than in three phenylketonuric children. However, the plasma indole and indican levels increased sharply in the phenylketonuric group, while the administered indole did not change at all the physiologic indole concentration in plasma of the controls. It was suggested by these authors that these findings are due to "a general derangement of the metabolism of indole compounds secondary to phenylalanine excess." In conclusion, it was thought possible "that the phenylalanine 'block' of tryptophan metabolism is the 'toxic' mechanism causing the mental deficiency." This speculation finds some support by the marked excretion of

TABLE II

Area as defined in test and illustrated in Fig. I. The tests in D.L. were conducted 5 months apart and in N.C. 2 months apart.

CONTROLS				PHENYLKETONURICS			
Name	Age	Sex	Area cm <sup>2</sup>	Name	Age	Sex	Area cm <sup>2</sup>
P.A.	16	m	68.7	D.L.	17	f	46.0
							42.4
S.W.	25	f	95.7	K.H.	17	m	6.4
B.V.	13	f	54.2	R.C.	15	f	19.6
P.H.	30	m	15.8	R.S.	16	m	27.0
G.S.	17	f	91.7	N.C.	24	f	62.0
							56.0*
A.G.	26	f	49.5	J.I.	28	f	87.5
Mean value			62.6	Mean value			43.4

\*This value was obtained while patient was on a diet low in phenylalanine. The phenylalanine concentration was 4.2 mg per 100 ml plasma and no phenylpyruvic acid was excreted.

indican and other tryptophan derivatives shown in another metabolic disorder causing mental deficiency, namely, in Hartnup disease, which is a familial condition characterized by aminoaciduria. However, no experimental evidence or other proof has been given so far that any tryptophan metabolite can cause mental deficiency. Therefore, the assumption of indican or any other tryptophan derivative as a causing agent for mental deficiency must still be regarded as a stimulating working hypothesis for further investigations.

An interesting abnormality in a baby girl with both phenylketonuria and the excretion of  $\alpha$ -hydroxy-butyric acid was described by Smith and Strang.<sup>71</sup> No quantitative data of the excretion of  $\alpha$ -hydroxy-butyric acid was given, since this compound had to be isolated from post mortem urine. The urine had an odor which was different from that usually found in phenylketonuric patients. Various observers described the odor of this girl as "like an oast house," like "burnt sugar," and "like dried celery." The authors believed that a double metabolic error existed in this child, which is most likely. However, since this child was not on a phenylalanine restricted diet, the possibility of a toxic effect of phenylalanine or its derivatives of  $\alpha$ -hydroxy-butyric acid metabolism was not excluded. The girl died at the age of 10 months.

When phenylpyruvic patients obtained a substantial oral dose of L-glutamine, the excretion of phenylpyruvic acid and phenyllactic acid could be reduced to about one-third of the control level. L-glutamate and L-asparagat were less effective. No effect was observed on phenylalanine of plasma or urine.<sup>62,82</sup> In contrast to glutamine and glutamate, the effect of asparagine on phenylpyruvic acid excretion occurred after a lag period. Meister, Udenfriend and Bessman,<sup>62</sup> who carried out these studies, explained the lag period as the time required to convert the inactive asparagine to the active glutamine.

Cawte<sup>20,21</sup> reported a significantly increased response to the Funkenstein test in phenylketonurics compared

with non-phenylketonuric controls. He measured the rise of the systolic blood pressure and the homeostasis time, following the intravenous injection of adrenalin. The homeostasis time was defined as "the number of seconds elapsing from the injection of adrenalin to the restoration of basal blood pressure." If rise in blood pressure and homeostasis time are plotted against each other on graph paper, the enclosed area can be calculated and this is the function of these two parameters. Although the rise in blood pressure did not show a significant difference between phenylketonurics and controls, the homeostasis time was 1.36 fold longer and the area 1.66 fold larger in the phenylketonurics. Cawte's co-worker, Mittwoch,<sup>21</sup> considered the difference of homeostasis time and area between the two groups as statistically significant. Following a suggestion by Knox and Hsia,<sup>55</sup> we examined the effect of adrenalin on blood sugar concentration.<sup>1</sup> Six untreated phenylketonuric patients ranging in age from 15 to 28 years were used for the test. Six mentally retarded patients, who otherwise did not demonstrate any physical abnormality, served as a control group.<sup>2</sup> The values obtained are summarized in Table II. No attempt was made to determine the significance of the mean values between the 2 groups in Table II, since the number of

<sup>1</sup> This preliminary study was supported by a PHS Research Grant No. 2729 from the National Institutes of Mental Health, Public Health Service. Credit is due to Mrs. C. Woodard and Miss Song Que Lee for determination of the blood sugar concentrations.

<sup>2</sup> The experiments were carried out in the morning, starting about 8 a.m., with breakfast being omitted. After taking a fingertip blood sample for blood sugar determination (baseline value), 1.0 mg of adrenalin per 70 kg body weight was injected subcutaneously in the upper part of the thigh (zero time). The blood sugar concentration was determined after 15, 30, 45, 60, 90, 120, and 150 minutes, and carried out in duplicate, according to the photometric micromethod of Folin and Malmros.<sup>26</sup> The values obtained were plotted on graph paper as mg glucose per 100 ml blood against time, and the enclosed area of these parameters was measured in cm.<sup>2</sup>

patients is too small. However, our data clearly demonstrate that no indication of an increased reaction in blood sugar concentration to adrenalin is present, as could have been assumed from Cawte's experiments in the Funkenstein test. Whether or not there is a tendency in the phenylketonuric group to be less sensitive to adrenalin remains to be proven on a larger scale.

#### PHENYLKETONURIA IN ANIMALS

##### (EXPERIMENTAL AND NON-EXPERIMENTAL)

A new approach for studying the effect of high phenylalanine concentrations on metabolic processes was opened by Auerbach, Waisman, and Wyckoff,<sup>6</sup> who fed a diet rich in DL- or L-phenylalanine to rats for a few weeks. A decreased phenylalanine hydroxylase activity resulted in the liver of these rats, as compared to rats receiving the same diet but without the addition of phenylalanine. L-tyrosine was also effective. Waisman and co-workers<sup>76,77,78</sup> reported also the effect of high phenylalanine concentrations on infant monkeys (Rhesus). Both the rats and the monkeys showed marked retardation in discrimination-learning, as compared to their respective controls. Rats maintained on a diet supplemented with phenylalanine and tyrosine showed a marked excretion of phenylpyruvic acid, coinciding with a decrease of 5-hydroxyindoleacetic acid in the urine. In plasma, the increase of phenylalanine was paralleled by a decrease of 5-hydroxytryptamine.<sup>45</sup> The authors attributed the disturbance of 5-hydroxyindole metabolism as secondary to the increase of phenylalanine, confirming previous reports<sup>8,67,68</sup> in human phenylketonuria. Low brain serotonin levels in rats on a high phenylalanine diet were published by Yuwiler and Louttit.<sup>84</sup>

A very interesting observation was made by Coleman<sup>22</sup> at the Jackson Memorial Laboratory in Bar Harbor. He studied the coat color of mice as to their mode of inheritance. The strains DBA/1J and DBA/2J were characterized by dilute pigmentation, extreme nervousness and seizures. These dilute mice had a diminished phenylalanine hydroxylase activity, ranging from 14% for the dilute, lethal strain (dl dl) to about 50% for the dilute, not lethal strain (dd) as compared to a not related and homozygous, nondilute strain, C57BR/cdJ (DD), while a DBA/1J (DD) strain had a hydroxylase activity of 86%. Evidence was obtained that an inhibitor of phenylalanine hydroxylase activity was present, possibly the phenylalanine derivative, phenylacetic acid. This substance was, namely, found to be present in increasing amounts with decreasing phenylalanine hydroxylase activities (DD  $\rightarrow$  Dd  $\rightarrow$  dd  $\rightarrow$  dl dl) in the nondilute and dilute strains. However, more proof will have to be provided before the assumption that a phenylalanine derivative might be the inhibiting factor is justified.

#### DETECTION OF HETEROZYGOUS CARRIERS

In 1954, on the basis of statistical analysis, Jervis<sup>47</sup>

suggested a single recessive autosomal gene for the phenylketonuric condition. If that is true, the parents of an afflicted child must both be heterozygous carriers of phenylketonuria. Hsia, Driscoll, Troll and Knox<sup>42</sup> introduced the phenylalanine tolerance test by which heterozygous carriers of this disease may be distinguished from any normal individuals or homozygous phenylketonuric carriers. This test is carried out in the same manner as an oral glucose tolerance test. After taking blood for the phenylalanine base line value (zero time) 0.1 g of L-phenylalanine per kg body weight are orally administered and the phenylalanine concentration of plasma is determined after 1, 2, and 4 hours. The heterozygous carriers show higher values than the controls above the already elevated fasting levels.<sup>41</sup> As Knox and Messinger<sup>56</sup> pointed out, a combination of both fasting levels and phenylalanine tolerance tests might allow a better classification of an individual into either the carrier or non-carrier group, since the values of both groups are overlapping and two criteria are superior to one. Hsia<sup>40</sup> recommended the phenylalanine-tyrosine ratio in the detection of the heterozygous carrier to be superior to phenylalanine levels alone in the fasting person. Jervis<sup>48</sup> has recently described a test using a dose of 0.33 g (2 mM) of phenylalanine per kg of body weight. When the plasma tyrosine concentration was measured photometrically with the 1-nitroso-2-naphthol method, a clear distinction between heterozygous, homozygous, and normal individuals could be made. Unfortunately, it has been our experience that the high doses of phenylalanine ingestion cause toxic side effects, such as dizziness and loss of appetite for more than 24 hours.

With increasing accuracy of the methods for phenylalanine determinations, the reliability of these tests will improve. Such tests might become of some importance for phenylketonuric individuals who, by treatment, will have normal intellectual development and consequently, because of their own experiences, would choose to avoid a heterozygous partner. Both tests have made evident the occurrence of a latent disturbance of phenylalanine metabolism in the heterozygous carrier for phenylketonuria. According to Tashian and Gartner,<sup>72</sup> who carried out phenylalanine tolerance tests with monozygotic and dizygotic twins, no evidence of a genetic control in the conversion of phenylalanine to tyrosine was obtained. However, the renal clearance of phenylalanine is controlled to some extent by genetic factors.

#### THE PREVENTION OF MENTAL DEFICIENCY

Clinical investigation in recent years has concentrated on the prevention of mental deficiency in phenylketonuria. Bickel, Gerrard, and Hickmans reported, in 1953 and 1954,<sup>13,14</sup> the influence of a low phenylalanine diet on a 2 year old girl who suffered from phenylpyruvic oligophrenia. When the diet was started in this child, the plasma phenylalanine concentration grad-

usually fell to normal levels and the excretion of phenylpyruvic acid ceased. The child's behavior improved during continued outpatient treatment; "she learned to crawl, to stand, and to climb on chairs, her eyes became brighter, her hair grew darker, and she no longer banged her head or cried continuously." On one occasion, without the mother's knowledge, phenylalanine was added to the diet. After a week the mother complained with distress that her daughter "had lost in a few days all the ground gained in the previous 10 months." However, in a later communication, Bickel, Boscott and Gerrard<sup>12</sup> reported that her growth in mental age did not keep up with her chronological age and that she remained on a low level.<sup>17</sup> Three other patients in whom this special diet was started at the ages of 3, 5, and 8 years were maintained on the diet from 3 months to 1 year, but no marked changes were noticed. The authors felt that a beneficial effect could only be expected when the diet was started before the age of 2 years.

These publications stimulated more work in this direction and reports by other authors soon followed.<sup>3,5,10,37,38,39,44,80,81</sup> The diet generally used is a casein hydrolysate from which most of the phenylalanine has been removed. Synthetic amino acid mixtures have also been given a trial;<sup>5</sup> however, these are too expensive for long-term treatment and offer no advantage to hydrolysates. The casein hydrolysate is essentially the sole source of protein nutrition and has, therefore, to be supplemented with carbohydrates, minerals and vitamins, since the intake of other food stuffs, such as vegetables and fruits, is restricted because of their phenylalanine content. Commercial preparations of casein hydrolysates<sup>1</sup> are now available. Acosta and Centerwall,<sup>1</sup> as well as Lyman and Lyman,<sup>59</sup> have recently published a list of menus using a greater variety in the meals in order to make available a more palatable diet.

Patients receiving this diet show a drastic reduction of the plasma phenylalanine to normal values over the period of a few weeks, and the phenylpyruvic acid excretion ceases after a few days to about a week. With the biochemical normalization, an improvement in behavior and physical condition, regardless of at what age the diet is started, has frequently been noticed. The patients seem to be more alert, less irritable, and the attention span is slightly increased. Of 14 patients with skin abnormalities at the beginning of the dietetic treatment studied by Hsia and co-workers,<sup>44</sup> 9 patients showed improvement of their skin condition. Darkening of hair has been reported in 11 cases out of 22 treated patients,<sup>44</sup> but not when an additional supply of tyrosine was given without a phenylalanine restricted

diet.<sup>61</sup> These *in vivo* findings are in good agreement with the already mentioned *in vitro* studies of tyrosinase inhibition by high phenylalanine concentrations.<sup>23,65</sup> The abnormal EEG pattern frequently seen in untreated phenylketonuric patients often changes to a more normal pattern by alimentary treatment,<sup>3,12,39,81</sup> but worsening was also reported in 2 cases.<sup>44</sup> Epileptic seizures frequently observed in phenylketonuric infants disappeared soon after the regimen was started.<sup>3,81,12</sup> On the other hand, changes from petit to grand mal have also been reported in 3 cases.<sup>5,12,81</sup> Knox and Hsia<sup>55</sup> reported a case which came to their attention where death occurred in status epilepticus after the initiation of a phenylalanine restricted diet, possibly associated with the rapid decrease in phenylalanine concentrations to near zero values. It should, therefore, be emphasized that adequate laboratory controls of the plasma phenylalanine concentration have to be carried out. Chemical controls which are limited to the phenylpyruvic acid content in urine are insufficient and might result in a retention of phenylalanine in plasma and tissue just below a concentration where phenylpyruvic acid is not yet excreted. We have observed such a case. The patient was first seen when he was 2 years old. Although he had been on a phenylalanine-restricted diet for about a year, his plasma phenylalanine concentration on one occasion was 21.8 mg per 100 ml, and he had made no significant intellectual progress during this period of time. After the adjustment of his phenylalanine concentration to about 2 mg per 100 ml plasma, a marked change of his mental development began. He learned to walk within a few weeks, became more alert and co-operative, and within 7 months his I.Q. increased from 35 to 49 on the Cattell Infant Intelligence Scale. Expressed in mental age, he advanced about 7.6 months. Thus, his mental age progressed at a slightly faster rate than his chronological age.

In a most thorough, 1 year controlled study of 29 treated cases at the Wrentham State School, Hsia and his associates<sup>44</sup> demonstrated that no intellectual improvement can be expected if treatment starts in older patients. By that time, the normal development of the brain has been disturbed to such an extent that irreparable damage has occurred. No definite age can be given when damage has become permanent, but it may safely be assumed that impairment of brain function in untreated phenylketonuric individuals starts immediately after birth. Woolf and co-workers<sup>81</sup> stated that mental deterioration occurs rapidly during the first few months of life and more slowly after the second year. However, this did not exclude the possibility that some reasonable progress for young children on a phenylalanine restricted diet might be expected. They noticed such an improvement in a girl of 2 years and 8 months of age with an I.Q. of 42 when the diet started. After continuous treatment for almost 3½ years, the I. Q. had gone up to 65 (case 3 of Woolf's

<sup>1</sup> Ketonil, Merck and Co.; Lofenalac, Mead Johnson Co. The latter company supplies, on request, a detailed description of the amount of food stuff allowed daily. This has, in our own experience, proved to be of value for good dietetic adjustment. However, an additional multi-vitamin supply in excess of that added to the casein hydrolysate is recommended.

observation, 1958). A similar case (case 2 of the same publication) showed an increase of her I. Q. from 71 when treatment was started at the chronological age of 3 years to an I. Q. of 92 at the age of 5 years. The latter observation is certainly an exception, since the patient already had a borderline intelligence at the time the diet was started. An opposite example was given by Bickel<sup>1</sup> who reported the case of a young school girl with an I. Q. of about 80 to 90, in whom dietetic treatment was not carried out, and her I. Q. subsequently dropped to significantly lower levels. These examples indicate the benefit which can be achieved with a phenylalanine restricted diet in young children who have a relatively high intelligence at the beginning of the regimen. Also, Horner<sup>39</sup> suggested, on the basis of his observations, that treatment of older phenylketonurics may prove valuable in some cases. But these are exceptions, as already mentioned. On the whole, no significant improvement can generally be expected in patients over 1 to 2 years of age.<sup>3,10,12,44</sup> This statement is, however, not meant to discourage the start of a treatment up to the age of 5 years if the intellectual potential is such that a permanent institutionalization might be spared or a decline of the intellect is avoided.

What dramatic changes might occur if treatment is started early in life, were observed by us in a boy who was first seen when 6 months 3 weeks of age. At that time the child obtained a mental age of 3.6 months, or an I. Q. of 54 on the Cattell; his social maturity age was 2.2 months. After treatment with Lofenalac for 10 weeks, his mental age had increased to 7.6 months on the Cattell giving him an I. Q. of 84, that is, his mental growth exceeded his chronological growth by about 7 weeks within this short time. On the Vineland Social Maturity Scale his mental growth doubled his chronological growth, and he obtained a S. M. Q. of 7 months. When the boy had reached a chronological age of almost 12 months he obtained on the Cattell a mental age of 9.8 months, and on the Vineland a S. M. Q. of 11 months. Now, at the age of 13 months, he is able to walk, to climb chairs, to climb stairs by himself. He also says a few words clearly and shows an absolutely normal development.

Another question of prime importance is the length of time the diet need be restricted. Armstrong<sup>3</sup> and Bickel,<sup>15</sup> for instance, believe that the diet may be discontinued after some years. Nevertheless, the time has not yet come to speculate in this direction, since long-term observations have not been made in sufficient numbers. Careful return to a more normal diet in later childhood under steady observation might give us some clue as to whether it will be possible to discontinue permanently a phenylalanine restricted diet. Since it is known that phenylketonuric patients with

high phenylalanine concentrations in plasma and tissue frequently present behavioral problems because of their irritability, this might, in itself, temper the choice of an absolutely normal diet.

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Editors Note: The Biochemical Laboratory of the Pineland Hospital & Training Center offers assistance in the dietary adjustment of phenylketonuric individuals. A short-time admission for dietary adjustment is now possible. For any questions concerning the treatment of phenylketonurics, contact H.-D. Grümer, M.D., Biochemical Laboratory, Pineland Hospital & Training Center, Pownal, Maine.

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*Continued on Page 21*

# Constitutional Sexual Precocity During Infancy\*

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The diagnosis of sexual precocity in the female during the first year of life may present a difficult diagnostic problem. Early activation of the pituitary gland is the most frequent type of sexual precocity in childhood, and according to Novak<sup>1</sup> at least 90% of all cases of sexual precocity are of this type. The ratio males to females is approximately 1:4. The onset is usually gradual with the development of extragonadal sexual characteristics, such as a growth spurt, beginning growth of pubic hair and of breast tissue. Skeletal maturation is far advanced and the 17-Ketosteroid excretion elevated, depending on the stage of development, but never exceeding the normal adult level. The external genitalia, clitoris and minor labia are undeveloped at the beginning, and the vaginal smear does not reveal characteristic estrogenic changes. The development is strictly isosexual and complete. Dental, psychosexual and mental development are consistent with chronological age. The further course follows the same pattern as in normal children, and sex development is completed within 2 to 2½ years, with onset of the menstrual period. Linear growth thereafter slows down and comes to an early standstill.

We had the opportunity of observing 2 infants, one at the age of 9 months, the other at the age of 15 months, who revealed a similar history and showed similar signs and symptoms on admission to the hospital. In both instances the course of development was entirely different from that in the older age group and its harmonious lawfulness characteristic of children beyond infancy was disrupted. They started, so to speak, from the other end, or at least, showed only very slight appearance of extragonadal development a few weeks prior to menstrual bleeding. Sex development is over precipitated at this early age, and the succession of its partial components condensed in time. The long interval between the early showing of extragonadal development and ovarian activity is missing. For this reason, such cases have to be more carefully investigated, as far as differential diagnosis is concerned, and the malignant types of sex precocity ruled out. Granulosa cell tumors, for instance, can be observed as early as at 5 months of age.

*Case I.* Nine month old white female. Two months prior to admission the parents first noted a slight fuzz of pubic hair. For the past 3 weeks they had noticed rounding of the

breasts. Five days prior to admission patient began vaginal bleeding which continued for 4 days.

Family History: Non contributory. Six siblings normal.

Physical Examination: A well developed, well nourished, white female, with slight extragonadal sexual characteristics. Fontanels are closed. Pupils and fundi within normal limits. Chest: clear to percussion and auscultation; there is breast tissue present bilaterally. Heart: normal sinus rhythm; P2 greater than A2; no murmurs are heard. Genitalia: there is only slight pubic hair present; clitoris is somewhat enlarged for her age. Labia minora are well formed. Neurological examination is negative.

X-ray of the wrist showed a bone age of approximately that of a 2 year old girl, in contrast to the chronological age of 9 months. X-ray of chest and skull were within normal limits. Intravenous pyelogram was normal. Two hour postprandial blood sugar, BUN, sedimentation rate, white count and differential count were all normal. Hematocrit 37%; Hemoglobin 10.6 gms.; Urine within normal limits. Calcium, phosphorus and alkaline phosphatase, and Micro Hinton, were all normal. Twenty-four hour urine for 17-Ketosteroid: 0.5 mgs. in 24 hours, 17-hydroxycorticosteroids: 1.06 mgs. in 24 hours. On pelvic examination under general anesthesia no masses were felt. Rectal examination revealed a small uterus in the midline with small ovaries; no evidence of any masses. Vaginal examination with a small nasal speculum revealed a normal vagina; no evidence of urogenital sinus. Twenty-four hour urine specimen for estrogens revealed 12 micrograms per 24 hours. This amount is insignificant. Study for FSH was negative. Smears of the vagina for cytology showed 65% precornified with occasional cornified cells; 35% intermediate cells. Smear showed evidence of estrogen activity, no malignant cells were seen.

The child was re-admitted at the age of 1-1/12 years for re-evaluation. A repeated pelvic examination under general anesthesia revealed no evidence of a mass or tumor or any abnormality on palpation. Cystoscopic examination of the vagina and cervical orifice revealed nothing remarkable. No evidence of foreign body was found.

Final Diagnosis: Constitutional sex precocity.

*Case II.* Fifteen month old white female, who was perfectly well until 1 month prior to admission, when the mother noted dark red mucoid vaginal discharge, on 3 occasions over 2 days, "like the end of a menstrual period." She had normal prenatal, natal and neonatal history. Normal growth and development.

Family History: Non contributory. No recent infections. Normal activity. On no medication, and no hormones taken by mother. There has been a question of recent breast development over the past month, noted by the mother, father, and referring physician.

Physical examination on admission revealed a temperature of 99.8, pulse 120, respiration 22. Weight: 25 lbs. 6 oz. (85th percentile). Height: 32 in. (90th percentile). Head circumference 19 in., chest circumference 19-1/2 in. Patient is a well developed, well nourished white female, in no acute distress. Head is normocephalic; the eyes showed no visual field defect. The pupils were round, regular and equal, re-acting to light. Fundi were normal. Extraocular movements were intact. Chest was symmetrical. Breasts showed bilateral increased tissue mass, with nipple formation and increased pigmentation. The

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lungs were clear throughout. Heart had normal sinus rhythm, with no murmur. Abdomen was soft, with no abnormal masses palpable. Genitalia: there was fresh red blood exuding from the vaginal orifice. Genitalia were normal for age. There was a fine blondish axillary hair distribution, and some fine pubic hair being present. Neurologic examination was negative.

Wrists for bone age not significantly abnormal for the given chronologic age. IVP was within normal limits, with no evidence of abnormal masses. Urinalysis normal. Micro Hinton was negative. Sed. rate was 3 (Landau and Westergren). BUN was 14. White blood cell count on admission was 16,500, with 52% neutrophils, 9% bands, 32% lymphocytes, 7% monocytes, 11.7 gms. hemoglobin, hematocrit 30%. Culture of the vaginal bleeding showed *B. aerogenes*, urine cultures repeatedly were negative. Throat culture showed alpha hem. strep. Urinary 17-Ketosteroids: 1.2 mg in 24 hours.

Vaginal smear showed 20% cornified cells and 80% per-cornified cells, which were indicative of increased estrogen activity compatible in amount with that of the early proliferative stage of a normal cycle. The pre-cornified cells showed slight folding, and contained a small amount of glycogen as usually found associated with slight progesterone activity as a carry-over effect from the previous cycle. No cancer cells were present.

Pelvic examination under anesthesia: the external genitalia appeared to be normal. The introitus was enlarged; the vagina was moderately well estrogenized, with marked rugae formation; the cervix was digitalized and blood was noted coming from the external uterine os; the uterus, on bimanual examination, was enlarged for the age. No adnexal masses were palpable. It was the impression that this was probably constitutional precocious puberty; cerebral disease was to be ruled out. Skull films were obtained, and showed no increased intracranial pressure, no abnormal calcifications or aberrations from the normal. After discharge from the hospital, she had another menstrual bleeding of 3 days duration after a 28 day interval.

On the occasion of a second hospital admission, an exploratory laparotomy was performed and biopsy material of both ovaries obtained. The ovaries grossly revealed to be moderately enlarged, each of them measuring approximately 2 x 1 cm. This enlargement was due primarily to one large single cyst within each ovary. The cysts were aspirated and biopsies of their walls obtained. Pathological report of the biopsy showed normal infantile ovarian tissue with no evidence of activity.

Both cases follow the same pattern. Only Case I could be observed over a period of 3 years. The second child moved out of town and no further contact with the family could be obtained. In both instances vaginal bleeding was the first noticeable sign of precipitated development and alerted the parents to have the child hospitalized. Traumatic etiology, foreign body, heman-gioma, accidental ingestion of stilbestrol containing material which was reported in several other cases could be ruled out. The first question was the possibility of an ovarian malignant tumor. However, the most common tumor, the granulosa cell tumor, is usually large enough to present itself on abdominal palpation. Nevertheless an explorative laparotomy is indicated in such patients, and was carried out in 1 of our 2 patients

with negative result. Adrenogenic etiology was easier to rule out. The absence of heterosexual characteristics, of an excessive output of 17-Ketosteroids, of deepening of the voice, of electrolyte imbalance, and of anomalies of the external genitalia did not favor this etiology. Neurogenic sexual precocity may simulate the constitutional type; but in both of our 2 patients, there were no suggestive neurological signs or symptoms, no increased intracranial pressure or abnormal calcifications on x-ray examination of the skull. In the male careful study is still more indicated, because of the relatively rarer incidence of constitutional sexual precocity.

By ruling out 3 of the 4 main types of sex precocity we were left with the diagnosis of constitutional early activation of the pituitary gland, in spite of the deviation from the usual pattern seen in later childhood.

In older children linear growth slows down considerably at the menarche and stops entirely shortly afterwards. If this occurred in infants, with the menarche in the first year of life, the final stature of the individual involved would be extremely short. However, our first patient did not follow this pattern. The growth rate during the first 3 years of life was rather satisfactory and showed the customary spurt. The yearly increments amounted to 4½ inches in the second, and to 3½ inches in the third year of life. At 2-9/12 years she was able to wear the dresses of her 5 year old sister. At the present time, at the age of 3-8/12 years, the height is 45 inches. At the same time breast development and pubic hair growth were slowly but steadily progressive. The menstrual periods were not quite regular. There were usually 2 in 28 day intervals, and cessation for 1 month thereafter.

It is hard to predict which height will finally be reached. There are no exact data available in the literature. It is probable that the normal increments in the early cases are the result of the high potential of growth in the first 3 years of life, and that the growth rate is not yet diminished by the female sex hormone. If this growth rate can be maintained up to school age, these children will not be worse off than other cases of constitutional sexual precocity with a later age of onset. It seems to be that at that early age the gonads are more sensitive to the effect of estrogen than in later childhood. On the other hand, skeletal development is much less advanced, in Case II it was even within normal limits which is different from the constitutional type of sexual precocity in children beyond infancy. Also, we would expect a much higher output of 17-Ketosteroids in girls after the menarche, in whom the pituitary-gonadal-adrenal mechanism is the same as that in normal children.

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# Are Human Milk Banks Still Necessary?

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Several decades ago human milk was considered not only *the* natural and ideal food for the normal young infant but was used in the treatment of the most severe manifestations of malnutrition in the young as the only sure and efficient dietary remedy. In chronic, recurrent diarrhea or in protracted under-nutrition with severe marasmus, Czerny and Finkelstein gave a feeding regimen with human milk as the only treatment likely to result in recovery and cure.

Even today in many technically underdeveloped countries with poor general hygiene, widespread intestinal infectious diseases and interfering taboos and superstitions, breast-feeding is the best and, in many parts of the world, the only insurance for the survival of young infants.

In the past three decades socio-economic factors and the uninterrupted remarkable progress of public health measures plus technological advances in the dairy industry have led, in this country, to a progressive decrease in the popularity of breast feeding. Today, human milk is not considered, any more, as an essential food for normal or malnourished infants.

Since the early Thirties, in this country, it has been rare to find human milk in the dietary of Nurseries or Children's Hospitals. Even premature infants were — and still are — reared, with very few exceptions, on cow's milk formulae whereas, even today in Western Europe, human milk with adequate protein supplements (cow's milk, casein, protein-hydrolysate etc.), forms the basis of the diet of premature infants.

Breast-feeding even for infants born at term became a rarity in this country. According to a recent statistical survey,<sup>1</sup> on the average only about 20% of the newborn infants are breast-fed at the time of their discharge from the Nursery.

From a teleologic point of view, human milk must be considered superior to cow's milk as the initial food for the human infant. In teleologic language: Human milk is for the human infant and cow's milk is for the calf. On the other hand, one has to admit that with good aseptic care it is difficult to do harm — or at least to cause demonstrable harm — to a normal infant kept on any feeding regimen which provides the minimum requirements for all essential nutrients. From a practical point of view, any formula type of infant feeding with adequate consideration of hygienic principles yields good results: Cow's milk formulae, mixed feeding, even at an early age — with vitamin supple-

ments will support satisfactory growth and development in a normal infant. A quotation from a recent report of the Committee on Nutrition of the Academy of Pediatrics summarizes the present status very well: "The practice produces neither beneficial nor harmful results but rather attests the adaptability of the baby to the whim of his caretakers."<sup>2</sup>

One of the distinctive features regarding infants fed human milk, as compared to those fed cow's milk refers to the intestinal flora and the reaction of the feces. In contrast to the acid reaction of the feces of normal breast-fed infants, the pH of the feces in infants given the usual cow's milk formulae falls in the neutral or alkaline range. Unlike the mixed intestinal flora of infants on cow's milk formulae, the intestinal flora of healthy breast-fed infants is characterized by the prevalence of a particular species of *Lactobacillus*, namely *Lactobacillus bifidus*.

The metabolic role and influence of the "Bifidus-Flora" in the normal breast-fed infant has been widely discussed in the old — and even in the more recent pediatric literature,<sup>3,4</sup> but it is still shrouded in mystery. The well-documented old observation on the change and gradual disappearance of the Bifidus Flora in breast-fed infants suffering from and even preceding not only intestinal but also parenteral infections seems to indicate a causal relation between "well-being" and "physiological" intestinal flora.

Scepticism against the overestimation of the differences in the intestinal flora between breast-fed and artificially fed infants is best countered — or at least shaken — by reference to the very interesting, not well-known disease-entity, familiarly named acrodermatitis enteropathica. The disease is characterized by vesicular dermatitis around the body orifices and the distal parts of the extremities, with multiple paronychia on hands and feet, diarrheal attacks and other digestive dysfunction.

The nature of this intriguing condition is unknown; it is probably based on an inborn metabolic error with its original site in the intestinal flora and, in further consequence, in the inability of the organism to detoxify bacterial products absorbed from the lumen of the intestine.

The disease usually appears after weaning and without treatment is usually fatal. Brandt,<sup>5</sup> in 1936, stated that — "Among all therapeutic experiments that have been instituted, the treatment with mother's milk is the only one that has any demonstrable effect. After addition of mother's milk to the diet there has been an increase in the baby's weight from a previously almost arrest of growth and, in particular, the general

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condition has shown a distinct improvement . . . Diet free from cow's milk or addition of goat's milk were ineffective."

More recently, the intestinal antiseptic Diodoquin® has proved to be a valuable remedy in the treatment of acrodermatitis enteropathica.<sup>9</sup>

It is of special interest that breast-feeding reduces both morbidity and mortality rates, especially the latter. Infants fed human milk — as compared with those fed cow's milk demonstrate higher resistance not only to intestinal disorders but to respiratory diseases including such complications as otitis media. Those of us, like myself, whose pediatric practice dates back many decades, will have no hesitation to testify in favor of breast-feeding in the prevention of infections. In this era, before the introduction of sulfa drugs, antibiotics and similar chemotherapeutic agents, human milk constituted the best *therapeutic* diet for infants with chronic and often very severe acute pyogenic infections. Similar observations were made in chronic intestinal disorders or severe malnutrition combined with chronic infection. In general, this effect of human milk in a variety of infections was ascribed not so much to the presence of specific antibodies but to the action of unspecific factors of unknown origin. Transfer of immune bodies from ingested human milk through the intestine into the blood is a negligible factor in resistance to disease if it occurs at all.

Improved general hygiene and the use of antimicrobial agents have, of late, obscured the superiority of human milk over cow's milk in studies of morbidity and mortality. Recent statistical studies, however, published from by no means under-developed countries like the United Kingdom and Sweden, indicate that these differences may be demonstrated not only under poor but also under good hygienic conditions.<sup>5-7</sup>

During recent years, well-documented personal observations were collected on the intriguing family epidemiology of viral diseases (common cold, influenza, mumps, coxsackie etc.). In many instances the infection attacked all or almost all members of the family, always including the mother but sparing the nursing baby. One example consists of a family with mumps affecting father, mother, grandmother and two siblings. The newborn continued to nurse while the mother had mumps but the breast-fed baby remained free from clinical manifestations. One could speculate that in this instance the infected mammary glands excreted with the milk not only virus-particles but also Interferon,<sup>10</sup> which in turn prevented the infection of the infant. This assumption is open to experimental verification since in this and all similar instances the mother herself had the viral infection. Diaplacental transfer of specific antibodies, which is usually operating in the resistance of young infants (up to 6 months or more) must be excluded.

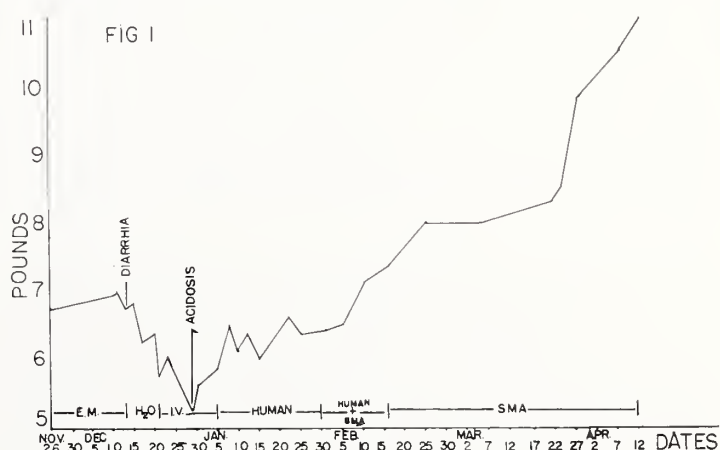
In addition to the prevention and treatment of infectious diseases or of chronic malnutrition combined

with infection, the use of human milk as temporary formula may be indicated and might even become indispensable in the treatment of severe forms of intolerance to cow's milk. True allergy to cow's milk, based on an underlying antigen antibody reaction is, in our experience, very rare.<sup>11</sup> In contrast, "intolerance" to cow's milk, often aggravated by inadequate dietary treatment of the condition is a fairly common observation in practice and may pose a difficult problem for successful management.<sup>12</sup>

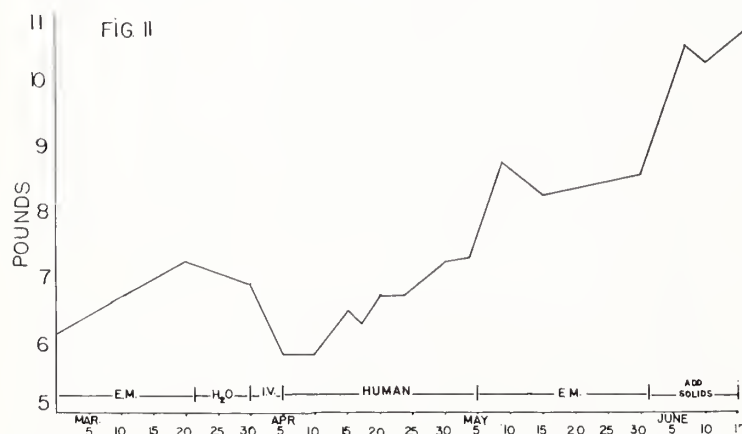
We have reported previously on a case of severe food intolerance, hypoproteinemia and generalized edema.<sup>13</sup> This 6 week old infant failed to thrive on evaporated milk mixture, soy-milk, and diluted cow's milk supplemented with Karo syrup or Dextri-Maltose.® No enzymatic difficulty was found after diligent search. When human milk was substituted he gained weight, became alert and happy and was maintained in positive nitrogen balance. The consecutive change to cow's milk was well-tolerated and has supported, with the usual dietary supplements, excellent and uninterrupted weight-gain.

During the past few years several observations were collected in our hospital on indications and use of human milk. A few pertinent examples are given below. The largest group comprises cases of severe, recurrent diarrhea, more or less resistant to the usual dietary treatment based on hydration and cow's milk-sugar-water formulae similar to the observations given above.

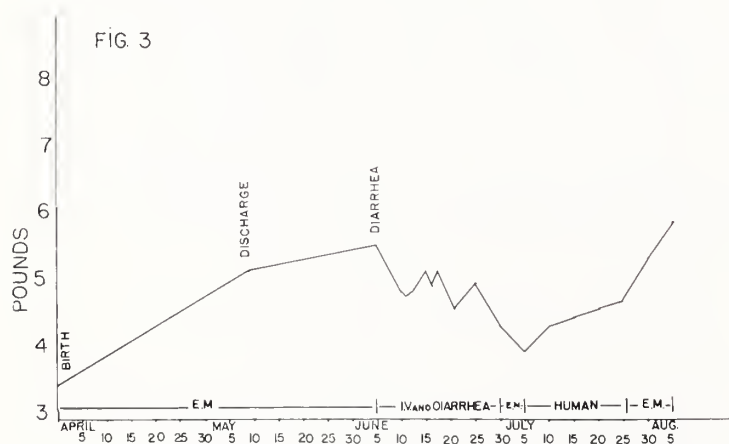
1. E.A., born 11-26-59, birthweight 2950 gms. Diarrhea developed at the age of 2 weeks (12-11-59) while the infant was taking a formula consisting of 1-part evaporated milk, 2-parts water with a 6% supplement of Dextri-Maltose #1. The infant was treated with the usual fluid regimen. On 12-21-59, he became moderately dehydrated and was put on i.v.-feeding with Saline/Glucose mixture for 3 days, and then oral feeding with Saline/Glucose and a small amount of Skim Milk. On 12-28-59, infant was very dehydrated with acidosis. Treatment consisted of whole blood (60 cc) i.v.-1/3 normal Saline with 2/3 Glucose (5%), and NaHCO<sub>3</sub> meq., for 16 hours. I.V.-Therapy was continued till 12-31-59. On 1-5-60, human milk was started with increasing amounts of 120-150 cc q 4 h. First slow, then rapid improvement was noted which continued after changing to a proprietary cow's milk formula (Fig. 1).



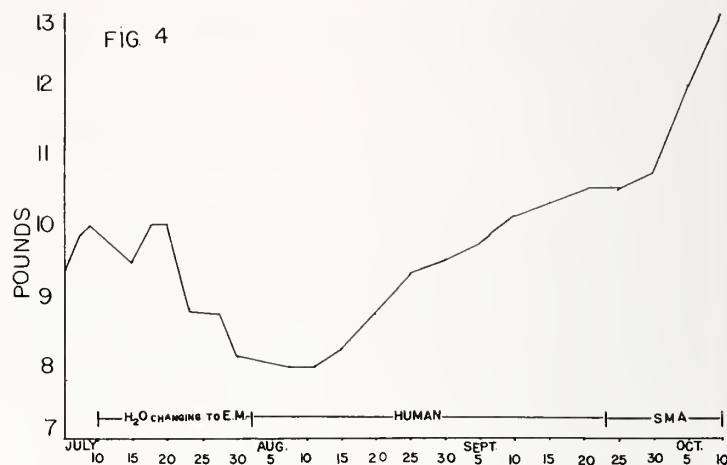
2. C.V., born 2-27-61. Birthweight 2750 gms. Baby was on an E.M.-Water-Sugar formula and developed diarrhea on 3-22-61. Treatment was begun with the usual anti-diarrhea regimen. Vomiting and diarrhea was noted on 3-31-61, which necessitated i.v.-therapy. Oral feedings were started on 4-1-61. More severe acute dehydration was then observed on 4-4-61. Treatment included intravenous administration of 1/4 normal Saline with 5% Glucose and 60 cc whole blood. Chloromycetin® 50 mg. q 8 h, because it was thought that she might have sepsis. Electrolytes were within normal limits. Human milk was started on 4-6-61. Baby was greatly improved after 2 days, diarrhea subsided and weight gain was resumed. Antibiotic was discontinued on 4-9-61. Feeding was changed to Evaporated Milk-Water-Sugar Formula on 5-4-61, followed with supplements of semi-solid food. Uninterrupted improvement (Fig. 2).



3. A.N., born 4-2-61. Birthweight 1600 gms. Admitted on 6-5-61, for mild diarrhea. I.V.-therapy and antidiarrheal regimen were introduced. Recurrence of diarrhea necessitated renewed anti-diarrhea treatment. Even on a regular Evaporated Milk-Water-Sugar Formula the infant continued to lose weight to 1800 gms. Regurgitation and poor activity complicated the poor clinical state. Electrolytes on 7-7-61, revealed Na meq. 120,  $\text{CO}_2$ , 7.7, Cl 107. I.V.,  $\text{NaHCO}_3$  corrected this condition. Human milk was started on 7-7-61, slowly increasing in amount. Rapid improvement followed which persisted on subsequent Evaporated Milk Formula (Fig. 3).



4. D.T., born 2-23-60. Birthweight 2650 gms. Admitted on 7-4-60, with mild diarrhea and marasmus. Weight 4200 gms. On anti-diarrhea regimen, diarrhea improved but weight-loss continued down to 3500 gms., on 8-7-61. Human milk was started on 8-4-61. Sharp, continued rise in weight followed which was maintained after change to a proprietary cow's milk formula (Fig. 4).



5. B.T., born 3-3-60. Birthweight 3100 gms. Admitted on 5-22-60, with right, middle lobe pneumonitis, left otitis media (draining) and multiple cutaneous pustules with *Staphylococcus Aureus*. Sensitive to Novobiocin and Chloromycetin. The infant was treated with Chloromycetin from 5-22-60, to 6-1-60, but pustules recurred. Human milk 120 cc x 6 q d was given from 5-24-60 to 6-20-60. No new skin lesions were noted after 6-4-60. All lesions were cleared on discharge, 6-20-60.

These few observations taken from a larger series should serve as illustration for the value of human milk in the dietary treatment of three groups of diseases in infants: a) Recurrent diarrhea, based often on "intolerance" to cow's milk. b) Marasmus and c) chronic, recurrent Staphylococcal (staphylococcal infections). Such clinical entities are by no means rare in our present day hospital practice. In consequence, need for human milk may often arise. Thus, human milk banks are still essential even in modern pediatrics. The human milk banks in Evanston, Illinois; San Francisco, California; Philadelphia, Pennsylvania; Wilmington, Delaware; Pittsburgh, Pennsylvania, etc., are fulfilling a very commendable and praiseworthy function. They provide human milk for our clinical practice but also, perhaps even more, they keep alive and spread with sincere zeal and enthusiasm the teaching of the psychological and nutritional importance of breast feeding among the young mothers of the United States.

Last but not least, their good deed will perhaps revive the interest of our pediatricians for the many problems of breast feeding, the scientific implications of which have been barely touched.

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## SYNOPSIS OF SOME CURRENT STUDIES IN PHENYLKETONURIA — *Continued from Page 15*

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DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

# Radioactive Fallout

ELMER W. CAMPBELL, DR. P.H.\*

A presentation of the problem of radioactive fallout is the subject under discussion in this paper.

The action of the University of Michigan Medical Center in officially adopting an emergency procedure for handling persons caught in radiation accidents, is a case in point. This procedure is by no means a war plan nor is it designed to handle a major nuclear disaster. It is simply a plan to establish a routine for handling a modern hazard. Under its terms, the basic treatment of patients will be directed by physicians and their clinical isotope unit. It will be the function of this unit to check the accident victim for residual radiation as well as to screen all hospital personnel who handle the patient. Hospital areas in which the patient has been treated will likewise need proper decontamination. In the plan, a provision is made for prompt, accurate news reporting to the public of all medical details of an accident compatible with the best interest of the patient.

Strictly medical aspects of the present level of radioactive fallout may very well be considered to be a matter of little or no immediate consequence. But its implications and what it may do if the fallout should increase in quantity is a matter that is not only worthy of much study, but one which is currently receiving a great deal of conjecture as to what the future may bring. For the moment, let us concern ourselves with the amount of radioactive iodine which is present in the daily fallout in this area . . . an amount to the extent of approximately 20%; or its relatively lesser amount of radioactive strontium, radioactive cesium, or even lesser amounts of radioactive carbon — all of which may have a bearing on this problem from a medical standpoint. These are problems for which a much more detailed discussion will be needed than can be undertaken at this time. However, we know the problem is there, and that it will receive much attention, — particularly if additional nuclear testing by foreign agencies takes place; or if we resume aerial nuclear testing, as well as underground testing, — such as has taken place within the last few days. This under-

ground testing has produced a small bit of radiation which was described as a cloud of steam with some radioactivity, and, at least insofar as the direction of the prevailing winds is concerned, did cause some unexpected radioactive fallout on the persons (and vehicles) of those in attendance who did not anticipate such exposure. The press releases from the agencies creating these tests have indicated that the amount was not excessive and was not considered dangerous but did constitute a decontamination problem.

Perhaps for the purpose of some basic understanding and review it would be well at this point to go back and define a few principles, as follows:

An atomic bomb, when it explodes, results in three effects in much the same manner as any other explosion, — namely, the release of light, heat, and blast or force. In addition, and at the same time there is the emission of nuclear radiation. This initial radiation is composed of neutrons, gamma and beta radiation, and lasts for only a short time, perhaps a minute, after the detonation. Beta radiation, because of its short path length, does not constitute any special hazard. Residual radiation is composed of gamma and beta rays from the fission products; alpha particles from the unfissioned uranium, or plutonium; and beta and gamma radiation from substances made radioactive by neutrons released at the time of the explosion.

When a ground burst occurs and dirt is sucked into the ascending cloud, vaporized fission products, or fragments, and neutron-induced radioactive elements, condense on this material. These contaminated particles (when they settle to the ground) are called fallout. High air-bursts do not produce significant fallout hazard because surface material is not carried into the cloud for the radioactive particles to condense upon. For the same reason, ground-bursts create a larger amount of radioactive particles which will then be carried up into the atmosphere and distributed through the upper currents of air — perhaps even around the earth.

An understanding of meteorological processes will help to create an understanding of the pattern of fallout distribution. Such processes have provided information about the rate of fallout debris and the manner in

\*Director, Division of Sanitary Engineering

which it reaches the earth's surface. Some of these substances appear to reach the lower atmosphere in two different ways. One, by the sinking of air in the polar regions in the winter; and the other by vertical diffusion or horizontal transport in middle latitudes. It would seem that in other places, diffusion downwards is impaired by the tropopause, — the surface of separation between the stratosphere and troposphere.\* However, in middle latitudes a gap in the tropopause is frequently present and through this gap the movement of debris is able to take place. For this reason, radioactive debris which is released into the stratosphere in high latitudes, particularly in the fall is transferred quickly to the lower atmosphere and deposited fairly rapidly on the surface of the earth. On the other hand, debris released into the stratosphere in tropical latitudes, may take considerable time, — perhaps months or years, — before it reaches or moves into higher latitudes from which it can be more readily transferred to the lower atmosphere.

Another factor that influences the stratospheric residence time is the size of nuclear explosions. This can be expected, since the more powerful the explosion the greater the height attained by the debris — a factor which may have a considerable effect upon the type of radioactive fallout. That which occurs quickly, as shortly after an explosion, may be expected to contain more of the short-lived products, — and that which is delayed for a long period of time, obviously would have a much lesser amount of the short-lived products. However, the longer-lived debris, such as strontium and cesium will still remain and be expected to come down at a much later period of time than that initially following an atomic explosion.

How then is this going to affect our outlook in regard to our activities relating to radioactive fallout? Those products which are of principal medical interest in radioactive fallout may be said to be: radioactive iodine 131, which has a half life of 8 days; radioactive strontium 89, with a half life of 53 days; radioactive strontium 90, with a half life of 27 years; and radioactive

cesium 137, with a half life of 27 years. Because of the short elapse of time since the nuclear testing by Russia, this fallout has been one which contains a relatively high percentage of iodine, which is short-lived material. Therefore, we can expect that a definite proportion, (perhaps approximately 20% of this fallout) was iodine 131. This may vary somewhat, — and unfortunately the necessary instruments to test this fact are not immediately available in this State so that outside sources must be utilized for the information desired in this respect at the present time.

For the same reason, it can be expected that fallout some months from now will have at least some increase of the strontium 90, because of its long life, and of the total amount of radiation found in fallout, a higher amount of it will be of these longer-lived portions than in the fallout currently present in our atmosphere that is being washed down with rain or snow.

It may be of general interest to know how and to what extent atmospheric tests are made by the Division of Sanitary Engineering of the Department. Samples for testing of the atmosphere are collected daily by means of an appropriate air pump located on top of the State House in Augusta. This pump runs continuously for 24 hours, pumping 75 to 80,000 cubic meters of air through a filter in a 24-hour period. The particulate material, which has collected on these filters, is then removed at a definite time each day and taken down to the laboratory where the preliminary readings are made. These preliminary readings are then recorded and publicized. Since the testing program began this past September, the readings have jumped up from a low of about 1/10 (0.1) micro microcuries per cubic meter to a maximum of 71.2, which is the highest preliminary reading yet recorded in this area. This, compared with a four day reading, — when it dropped to 37.5, — indicates how quickly the radioactivity decreases. The fluctuations, however, from day to day have been measurable, and have been consistent with those which have been found in Massachusetts. There appears to be about the same amount of radioactivity coming here, for instance, as at Lawrence, where the well-known experiment station is located. However, the amount of activity is quite different from that in Connecticut and further south. It will be of interest to learn that readings of more than four and five hundred were recorded in the middle states, — around Arkansas and in that general vicinity down through Tennessee and South Carolina, — following Hurricane Carla which apparently sucked in polar air with its attendant radioactive debris and permitted such debris to be deposited in that area at that time. No particular concern was felt as result of that activity, because at no time did it reach a gross beta activity micro microcuries per cubic meter of air of 1000 or more, — one of the limits provided. It would appear desirable here to present a table which has been prepared by the

Continued on Page 26

Atmosphere terminology.

Troposphere — Up to 25,000 ft. Temperature drops with or approximately increased height.

Tropopause — 25,000-60,000 ft. (5-12 miles) Ozone appears in small quantity at low levels then increases to a maximum at 65,000 ft., then decreases.

Stratosphere — Several miles Constant temperature, or increases with height.

Mesosphere — 15-50 miles Temperature increases first with height to a maximum (the Mesopeak), then decreases to a minimum (the Mesopause).

Thermosphere . . . . . Temperature increases with height.

a—Ionosphere 50 to 250 miles, high in ion density in the lower part.

b—Exosphere Above 400-600 miles the outer region, molecules escape from the atmosphere.

# County Society Notes

## PENOBSCOT

November 21, 1961

A meeting of the Penobscot County Medical Society was held at the Tarratine Club in Bangor, Maine on November 21, 1961. There were forty members and guests present.

Richard C. Wadsworth, M.D., President, presided at the meeting which followed a social hour and dinner.

Emerson H. Drake, M.D. of Portland, Maine, the speaker of the evening, gave a most interesting talk on "Cardiovascular Surgery" in which he traced the origin and development of the service at the Maine Medical Center. Dr. Drake's talk was complemented by a short presentation of other aspects of the subject by Peter Rand, M.D., Research Fellow at the Maine Medical Center. Following a discussion period, a rising vote of thanks was given to Drs. Drake and Rand for a most interesting and stimulating presentation.

H. Draper Warren, M.D. was elected to membership in the society at the business meeting which followed the Scientific Program. Brief interim reports were made by Edward J. Hughes, Jr., M.D. of the Diabetes Committee and by Byron V. Whitney, M.D. of the Public Relations Committee.

Communications in regard to 1962 State and National dues and the availability of reprints for patient distribution of the Graham Hutton article "America, Beware of the Welfare State" were read.

PHILIP B. THOMAS, M.D.  
*Secretary*

## WASHINGTON

December 7, 1961

A regular meeting of the Washington County Medical Society was held at the Congregational Vestry, East Machias, Maine on December 7, 1961. There were seventeen members and guests present.

The following officers were elected for 1962:

President, Rowland B. French, M.D., Eastport  
Vice-President, L. W. Brownrigg, M.D., St. Stephen, N.B.  
Secretary-Treasurer, Karl V. Larson, M.D., East Machias  
Delegate to the Maine Medical Association House of Delegates: Samuel R. Webber, M.D., Calais. Alternate: Hazen C. Mitchell, M.D., Calais

Board of Censors: John Kazutow, M.D., Ellsworth (3 yrs.); DaCosta F. Bennet, M.D., Lubec (2 yrs.) and William C. Rice, M.D., Calais (1 yr.)

Hadley Parrot, M.D. of Bangor, Maine spoke on "Chemotherapeutic Agents in the Treatment of Cancer."

KARL V. LARSON, M.D.  
*Secretary*

## HANCOCK

December 13, 1961

A meeting of the Hancock County Medical Society was held at the Hancock House, Ellsworth, Maine on December 13, 1961.

The treasurer's report was read and approved. The nominating committee consisting of Elizabeth E. Williamson, M.D., Llewellyn W. Cooper, M.D. and Arthur M. Joost, Jr., M.D. was appointed and presented the following slate of officers who were elected for the ensuing year:

President, James H. Crowe, M.D., Ellsworth  
Vice-President, Russell M. Lane, M.D., Blue Hill  
Secretary-Treasurer, Russell G. Williamson, M.D., Blue Hill  
Delegates to the Maine Medical Association House of

Delegates: Llewellyn W. Cooper, M.D., Bar Harbor and Elizabeth E. Williamson, M.D., Blue Hill. Alternates: Arthur M. Joost, Jr., M.D., Bucksport and Philip L. Gray, M.D., Blue Hill

Censors: Robert F. Russell, M.D., Penobscot (3 yrs.); Bradley E. Brownlow, M.D., Blue Hill (2 yrs.) and Elizabeth E. Williamson, M.D., Blue Hill (1 yr.)

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## KENNEBEC

December 14, 1961

The Kennebec County Medical Association met at the Augusta State Hospital in Augusta, Maine on December 14, 1961.

Joseph P. Senenkyj, M.D. of Augusta was elected to membership.

The following officers were elected for the coming year:

President, Loring W. Pratt, M.D., Waterville  
Vice-President, Brinton T. Darlington, M.D., Augusta  
Secretary-Treasurer, Earle M. Davis, M.D., Waterville  
Councilors, George J. Robertson, M.D., Waterville; Hugh J. Mathews, Jr., M.D., Gardiner and Allan J. Stinchfield, M.D., Augusta

Grievance Committee, William N. Runyon, M.D., Augusta; Hugh J. Mathews, Jr., M.D., Gardiner and John F. Reynolds, M.D., Waterville

William E. Schumacher, M.D., Director of Mental Health, Department of Health and Welfare, State of Maine, spoke on "Progress of the Mental Health Program."

EARLE M. DAVIS, M.D.  
*Secretary*

## LINCOLN-SAGADAHOC

December 19, 1961

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges, Wiscasset, Maine on December 19, 1961. There were nineteen members and guests present.

A nominating committee was appointed to bring in a slate of officers for the coming year.

Stanley C. Beckerman, M.D. of Waterville, Maine spoke on "Cancer Chemotherapy."

GEORGE W. BOSTWICK, M.D.  
*Secretary*

## CUMBERLAND

December 21, 1961

A meeting of the Cumberland County Medical Society was held at Valle's Steak House in Portland, Maine on December 21, 1961. A social hour preceded a dinner and business meeting.

A. Dewey Richards, M.D. was elected to membership in the society.

Officers elected for the coming year are as follows:

President, Robinson L. Bidwell, M.D., Portland  
Vice-President, Philip P. Thompson, Jr., M.D., Portland  
Delegates to the Maine Medical Association House of Delegates for two years: Charles R. Glassmire, M.D.; David K. Lovely, M.D. and Robinson L. Bidwell, M.D.

*Continued on Page 26*



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1. Sollmann, T.: A Manual of Pharmacology and Its Applications to Therapeutics and Toxicology, ed. 8, Philadelphia, W. B. Saunders Company, 1957, p. 206.

COUNTY SOCIETY NOTES — *Continued from Page 24*

of Portland and Robert H. Pawle, M.D. of Falmouth. Alternates for two years: George O. Chase, M.D.; John F. Gibbons, M.D. and Donald P. Cole, M.D. of Portland and Ronald A. Bettle, M.D. of Brunswick. Public Relations and Grievance Committee: Warren C. Baldwin, M.D., Chairman and Edward G. Asherman, M.D. of Portland and William F. Taylor, M.D. of Falmouth Foreside  
It was announced by the President that in January there

would be a joint meeting with the Woman's Auxiliary. A film entitled "On Call to the Nation" covering ten years of socialized medicine in England will be shown.  
Mr. Richard Dyke of the U.S. Internal Revenue Service was the guest speaker. He presented a film on income tax procedures and processing and supplemented this by a talk and question and answer period.  
ALBERT ARANSON, M.D.  
*Secretary*

DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page 23*

Federal Radiation Council on recommendations relating to various amounts of radioactive fallout when it has been reduced to known substances. Previous information has indicated that iodine was one of the items which would be of greatest concern initially and that about 20% of the present fallout may be considered to be iodine 131. There are not immediately available any proportions for other ingredients. This table will, however, serve to indicate the actions which are now contemplated when or if radioactive fallout should ever reach any levels where further action other than routine surveillance may be needed.

RANGES OF DAILY INTAKE (Micromicrocuries Per Day Averaged for 1 Year) FOR USE IN GRADED SCALES OF ACTION			
Radionuclide	Range I*	Range II**	Range III***
Iodine-131	0-10	10-100	100-1,000
Strontium-90	0-20	20-200	200-2,000
Strontium-89	0-200	200-2,000	2,000-20,000

Graded Scales of Action  
\* Range I Routine surveillance  
\*\* Range II Detailed surveillance and analysis  
\*\*\* Range III Consideration of control measures designed to limit intake

When radiation levels being detected in a particular area are in Range II, the action needed is to determine precisely how much and what kind of radioactive substances are actually likely to enter the human body over the course of a year by various routes, i.e., air, water, milk, and other foods. Federal, State and local governments are now making such determinations.

In general, as to the levels of radioactive fallout that we are concerned with in the State of Maine, a notation of the maximum and minimum amounts would appear to be adequate for present understanding. These vary from a low of about 0.1 (1/10) micro microcuries per cubic meter of air, to a maximum of 71.2. If 20% of this is iodine-131, it is probable that the iodine content would not exceed 15 micro microcuries per cubic meter. From the table above it may then be seen that this is in the Range II level, indicating that while it may necessitate detailed surveillance and analysis, it is not a situation to warrant any additional action at this time. It should be noted further that this maximum occurred only one day, and that the average for this period is 10.8, which is in Range I or the low range — requiring no further activities than routine surveillance. The strontium-90 content will be even less and much of this may be delayed even months before it will show up in any appreciable amounts. This then is a question for further study and evaluation, and, as has already been noted would appear to need a review at a later date, as the present conditions do not seem to require any additional action.

It may be anticipated that, if testing is resumed or anything occurs to change the present conditions or increase fallout, the National Surveillance Council, through the U. S. Public Health Service and other official agencies, will publicize detailed information for the guidance of public health agencies, hospitals, physicians and other key personnel.

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# The Journal of the Maine Medical Association

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No. 2

## The Diagnosis And Treatment Of Nasopharyngeal Conditions\*

FREDERICK T. HILL, M.D.\*\*

The nasopharynx is probably the most neglected region in the field of oto-rhino-laryngology. This may be due to its relative inaccessibility, behind the soft palate, and the frequent difficulty attending inspection. Yet the answers to many diagnostic problems may be found in the nasopharynx. The importance of thorough and careful examination of this region cannot be over-emphasized. As successful surgical procedures in this area require an adequate knowledge of the applied anatomy, this will be reviewed in the lecture.

### ANATOMY

Situated between the posterior choanae of the nasal cavities and the level of the free margin of the soft palate, the nasopharynx has been referred to by Carmody as "the crossroads of the respiratory and digestive tracts." It is encircled by the superior constrictor of the pharynx, attached at the median raphe and the pharyngeal tubercle. Its lower lateral boundary is the palato-pharyngeal arch, a fold of mucous membrane enclosing the palato-pharyngeus muscle. Contraction of these muscles, together with elevation of the soft palate, closes the nasopharynx from the oropharynx.

The mucosa is a continuation of the nasal mucous membrane. It is squamous cell epithelium except immediately posterior to the nasal cavities. It contains many mucous glands.

The blood supply of the nasopharynx is from the external carotid mainly by way of the ascending pharyngeal artery. There is a rich plexus of veins which empty into the pharyngeal vein and thence to the internal jugular. The lymphatics are very abundant and drain into the superior deep cervical and the retropharyngeal nodes.

The nerve supply is from the internal maxillary branch of the fifth, the pharyngeal branch of the ninth, and the pharyngeal branch of the tenth cranial nerves.

The orifices of the Eustachian tubes are found on the lateral walls, at about the level of the posterior ends of the inferior turbinates. Each orifice is bounded above and behind by a cartilaginous prominence, the torus tubae. Running downward from this is a fold of mucous membrane containing the rudimentary salpingo-pharyngeus muscle. Behind the prominence of the tubal orifice there is a deep recess, the fossa of Rosenmüller. Lymphoid tissue is often found filling these fossae. Lymphoid tissue may also be found within the tubal orifices.

The major portion of the lymphoid tissue, the adenoids, will be found in the upper posterior part of the nasopharynx. This tends to atrophy and disappear after the tenth year but may persist in varying degree through adult life.

Sometimes an opening may be found in the midline of the vault leading upward and backward to a cavity called the pharyngeal bursa, a remnant of the hypophysis cerebri.

\*Read before the American Academy of General Practice, Miami Beach, Florida, April 20, 1961

\*\*Thayer Hospital, Waterville, Maine

### EXAMINATION

The nasopharynx is not easily accessible to visual examination and as a consequence is not always accorded the thorough and complete inspection so essential to a correct diagnosis. Examination with the post-nasal mirror has definite limitations. The patient may be uncooperative or may possess an extremely sensitive throat. These difficulties usually may be overcome with patience and perseverance, although sometimes a local anesthetic may be required. There is often considerable variation in the contour of the nasopharynx, and it may be difficult to thoroughly inspect this region by means of the mirror. There is also more or less distortion of the image in any mirror held at a right angle to the object being examined. This may be further increased by the presence of mucous in the nasopharynx. In the hands of the experienced, however, this is quite satisfactory for routine examination.

The nasopharyngoscope possesses many advantages and may uncover conditions which were not recognized by mirror examination. In small children an antroscope, being of lesser caliber, may be used more easily. It is necessary to use the nasopharyngoscope through both sides of the nose so that the blind area directly ahead of the instrument can be included. Oftentimes the pharyngoscope used through the mouth is useful.

Anterior rhinoscopy, after shrinking the nose, will often give a good view of the superior-posterior part of the vault.

Palpation is an old and accepted method of examination but also has decided limitations. It is uncomfortable for the patient and is necessarily done so hurriedly as to be rather unsatisfactory to the examiner. Aside from determining the presence of adenoids or ascertaining the firmness of a tumor mass, it gives comparatively little information.

Direct inspection by elevation of the soft palate is probably the most satisfactory method of finally determining the condition of the nasopharynx. This is not suggested as a routine procedure but to be used when other measures fail to allow a thorough inspection. The Yankhauer speculum is very satisfactory, especially for viewing the lateral regions, such as the fossae of Rosenmüller. The Love palate retractor is a most useful instrument in many instances. In most cases the author finds his own palate retractor, a simple spatula, with the distal end turned at a right angle, the most effective instrument. Usually a minimum amount of local anesthesia in the adult is advisable. Small children may be restrained by being firmly wrapped in a blanket. By direct inspection one can see the actual picture without distortion and can better interpret the condition seen with the nasopharyngoscope or the mirror.

Examination should not be considered complete until the posterior choanae, the posterior margin of the vomer, the tubal orifices and fossae of Rosenmüller and the entire vault of the nasopharynx have been visualized.

The roentgenogram may be a most useful adjunct to visual examination and should be employed in all cases of suspected malignancy.

### ADENOIDS

The major portion of lymphoid tissue in the nasopharynx is found in the superior-posterior portion of the vault. It varies greatly in size and usually tends to atrophy and disappear after the tenth year.

Symptoms indicating surgical removal are nasal obstruction, chronic sinus involvement, chronic or recurrent otitis media, and conduction deafness. This latter may be first manifested by a drop in the upper tonal range, well above the speech frequencies. In adults a small residual adenoid may harbor infection and give rise to many symptoms referable to the nose, pharynx, and middle ear. Adenoid tissue in the fossae of Rosenmüller or adhesive bands across the fossae may result in tubo-tympanic conditions.

Adenoidectomy, too often, is poorly and incompletely performed. Tags of lymphoid tissue may be left in the vault or in the fossae of Rosenmüller. Sometimes these may present crypts sealed with scar tissue. These remnants may give rise to quite as much trouble, aside from mechanical obstruction, as the original adenoid. There is an unfortunate tendency to consider adenoidectomy as merely an adjunct to tonsillectomy. Actually an adequate adenoidectomy is more difficult to achieve than a good tonsillectomy. Visual inspection of the nasopharynx by elevation of the soft palate will greatly facilitate a clean operation. The finger may be used to dislocate the lateral portion of the adenoid from the fossa of Rosenmüller after which the mass is removed, using adenotome, curette, and punch cutting forceps, under direct visual control.

The removal of a residual adenoid in an adult is likewise done by direct inspection. In the same manner lymphoid tissue may be removed from the fossae and lateral walls, and adhesive bands destroyed. Electrocoagulation is an effective method in many instances. The value of irradiation of nasopharyngeal lymphoid tissue is debatable.

### HYPERTROPHY OF POSTERIOR TIPS OF INFERIOR TURBINATES

Hypertrophy of the posterior tips of the inferior turbinates may result in symptoms of conduction deafness as well as nasal obstruction. Often these are allergic in origin or may be associated with a chronic sinusitis. While sometimes they may be reduced by electrocoagulation, removal by snare through the nose is most effective. This may be facilitated by first injecting novocain into the hypertrophied tip.

### CHOANAL POLYPI

Posterior choanal polypi must be differentiated from nasopharyngeal fibroma. They are generally secondary to chronic sinusitis and usually originate in the antra,

although myxomata may be found attached at the posterior choanal or lateral walls of the nasopharynx.

#### FOREIGN BODIES

Foreign bodies may sometimes become lodged in the nasopharynx in infants and small children. These may be pushed back through the nose, or, more commonly, be regurgitated after aspiration or swallowing. Roentgenograms of small children, in which foreign bodies are suspected, should include the nasopharynx.

#### CONGENITAL ATRESIA OF THE POSTERIOR CHOANAE

Characterized by nasal obstruction and difficulty in nursing, the condition may be either unilateral or bilateral. Nasal secretion is thick and profuse. Cases in which the obstruction is largely membranous can be dealt with effectively by electro-coagulation. When the obstruction is predominantly osseous it is best to remove it surgically, together with a small piece of the posterior edge of the septum. The palatal approach has many advantages.

Occasionally one sees the reverse of the above, in which there is an absence of the posterior part of the septum due to lack of development of the vomer. These cases are rather prone to tubo-tympanic conditions but do not have speech difficulties provided the soft palate is normally developed.

#### NASOPHARYNGEAL CYSTS

The usual nasopharyngeal cyst is found in the midline of the upper part of the vault. These are of two types: The retention cyst in a residual adenoid, where a crypt has been sealed by inflammatory or scar tissue, and the cyst, considered by Dorrance as due to the persistence of an embryonal bursa, originating from adhesions of the notochord to the pharyngeal ectoderm. The retention cyst is easily opened with a probe, while the bursa will resist other than a cutting instrument. The latter appears smooth, symmetrical and from 1 to 2 cm in diameter. It may have a small opening in its center, either exuding secretion or covered with a scab. It is usually tense and fluctant, and contains thick gelatinous material. Either type of cyst may give rise to a post-nasal discharge, cause a variety of symptoms affecting the ear, or act as a focus of infection. They are best diagnosed and operated upon by direct inspection.

Occasionally one may encounter retention cysts, originating in the mucous glands of the nasopharynx. These are more apt to be on the lateral walls and may become large enough to mechanically obstruct the posterior choanae. They must be differentiated from nasopharyngeal fibromata, neuro-fibromata, etc. Removal is best effected by direct approach by elevation of the palate.

#### NASOPHARYNGEAL FIBROMA

More properly termed "angio-fibroma" or "vascular fibroma" because of their marked vascularity. While histologically benign, they are clinically malignant. They

are found predominantly in males between the ages of 7 and 25 years. They originate from any part of the fibrous tissues of the nasopharynx, usually from the periosteum of the basilar process of the occipital bone and the body of the sphenoid bone. They may invade the sinuses, orbit, sphenomaxillary fossa or the brain cavity. Their attachment is usually sessile and broad. Spontaneous involution and disappearance may take place at about the age of 25 years.

The tumor appears reddish, firm, smooth or lobulated. It often extends forward into the nasal cavity. The presenting symptom is nasal obstruction. Spontaneous hemorrhages are apt to occur. As the tumor grows it may involve the sinuses and the orbit and result in external nasal deformity. One cause of the author's had extended through the cribriform plate causing a pneumatocele within the anterior cranial fossa. Because of the tendency to hemorrhage, routine biopsy is not advisable. This is rarely necessary for the experienced examiner.

Radiation therapy with radon implants, together with electrocoagulation, is the treatment of choice in most instances. Some cases, especially if pedunculated, may be amenable to surgical removal but the surgeon must be prepared to deal with severe hemorrhage. Splitting the soft palate may afford adequate approach and the tumor may be evulsed by blunt dissection. Often the electro-coagulation wire snare is effective, in removal.

#### MALIGNANT NEOPLASMS

Malignant lesions of the nasopharynx may be overlooked until severe complications, or metastases call attention to this area.

Squamous cell carcinoma most commonly originates from Rosenmüller's fossa but may develop from within the eustachian tube. They tend to extend upward, invading the base of the skull and resulting in involvement of the cranial nerves; and may escape detection until the development of extra-nasopharyngeal signs and symptoms, such as cervical adenitis, pain, unilateral conduction deafness, diplopia, or disturbances in swallowing.

Lymphoepithelioma, and transitional-cell carcinoma tend to be unilateral and may result in tubal occlusion with conduction type deafness. There is a marked cervical adenopathy. In the early stage this may be confused with simple adenoid tissue. In operation for recurrent adenoids histopathologic examination should always be carried out.

Giant follicular lymphoblastoma, plasmocytoma, and lymphosarcoma may be encountered in the nasopharynx.

Sarcoma usually originate from the superior-posterior wall of the nasopharynx. They grow rapidly, produce early signs of nasal obstruction and tend to distant metastases. Hemorrhage is a frequent symptom.

Terratoma are occasionally encountered in the nasopharynx. Craniopharyngioma may develop from remnants of the hypophyseal duct. Chordomata, having

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# Assessment Of Vision Of The Pre-Literate Child

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While interest in the vision of the children of school age has increased recently and school vision testing programs are proving to be effective in detecting heretofore unrecognized defects, there are still numerous instances in which a defect is discovered too late for satisfactory correction. The importance, therefore, of testing the vision of children at the earliest possible age has been stressed by Fink,<sup>1</sup> Gundersen,<sup>2</sup> and Allen,<sup>3,4</sup> the latter having developed a vision test for pre-school children (approx. 3-5 years).

Physicians in general and pediatric practice have the greatest opportunity to test the vision of infants and young children and thus discover potentially remediable ocular defects. As further encouragement and assistance to these physicians in this worthwhile examination, the present paper summarizes techniques useful in the assessment of vision of the pre-literate child.

The development of vision is a complex process of intricate anatomic and neurophysiologic changes which is not complete until well after birth. The differentiation of the foveal cones continues through the first one to four months of postnatal life; medullation of the optic nerve fibers is completed in the first three weeks and other developmental changes in the iris, ciliary body and lens occur throughout the early years of life. To what extent the visual mechanisms of the cerebral cortex develop postnatally is not known but recent electroencephalographic evidence suggests that such development does occur at least up to the age of one year.<sup>5</sup> As these various changes occur the eye becomes more efficient as measured by the higher visual acuity which children normally achieve as they get older.

## AT BIRTH

Inspection of the eyes of the newborn will reveal any obvious abnormalities of the ocular coats or refractive media. The assessment of the visual mechanism may then be extended to include the following:

*Optical Blinking Reflex:* When a bright light is shone into the eye, the lids involuntarily squeeze shut (blepharospasm). This response is normally present at birth and indicates a functioning retina and optic nerve but does not indicate the degree of function, since the reflex has been elicited in infants with extensive retinal disease. The absence of blepharospasm in response to light in adults with no intrinsic disease of the pre-geniculate optic pathway has been taken to in-

dicate defects of the visual cortex. The preservation of this reflex in hydranencephalic children with no histologically demonstrable cerebral cortex suggests that in these infants, the pathway for blepharospasm in response to light is subcortical.<sup>11</sup> Whether this holds true for infants with other lesions of the visual cortex is not established.

*Direct and Consensual Pupillary Reactions to Light:* These reflexes are normally present at birth and depend upon a functioning afferent pregeniculate optic pathway (i.e. retina, optic nerve, optic tract). Absence of pupillary constriction when the eye is directly stimulated by light and preservation of the pupillary reaction of this same eye when the fellow eye is stimulated indicates non-function of this pathway.

Lesser lesions of the pregeniculate pathway may be detected at birth by a refinement of the test for pupillary light reaction, described by Marcus Gunn. In this test the right eye is illuminated while the left eye is covered; then the left eye is illuminated while the right eye is covered. If one pupil dilates while exposed to the light it indicates a lesion, most probably of the nerve fibers. A variation of the above is the so-called pseudo-anisocoria test in which the size of the pupil with the fellow eye covered is measured and compared with the similar measurement of the other pupil. The illumination for this test must, of course, be diffuse and fall equally on each eye. Under such circumstances, a definite difference in the pupillary size indicates a lesion on the side of the large pupil.

*Vestibular Reaction:* During rotation of the infant's head, the eyes normally deviate in a movement which represents the slow phase of vestibular nystagmus. The fast, or compensatory, phase is not directly dependent upon the stimulus to the vestibular apparatus and is not normally present until a few weeks after birth. Cogan<sup>6</sup> suggests that a convenient way of eliciting the vestibular reaction in infants is to hold the child in an upright position over and facing one's head and pivot quickly to right or left, observing the ocular movements during rotation. Normally, the infant's eyes will look in the direction of motion (slow phase of the reaction). If the fast phase is also present, it will be seen as a jerky movement of the eyes in the opposite direction. Thus, an estimate of the ocular motility, as well as the vestibular apparatus, can be obtained early in life. The value of this simple test of ocular motor function, particularly since it may indicate the state of the VI cranial nerves, is obvious.

*Opticokinetic nystagmus (OKN):* This is another response by which the vision of infants can be as-

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TABLE I

ASSESSMENT OF THE NORMAL VISUAL MECHANISM  
OF PRE-LITERATE CHILDREN

Age	Visual Reflexes as Noted by Cogan, <sup>6</sup> Kestenbaum, <sup>7</sup> Gorman <sup>8</sup>		Visual Acuity as Determined By		Allen <sup>3</sup>
			Schwartzing <sup>9</sup>	Chavasse <sup>10</sup>	
Birth	Optical Blinking Reflex Pupillary Light Reflexes Vestibular Reaction (slow phase) Opticokinetic Nystagmus (V.A. = 20/670)				
2-4 weeks	Optically Elicited Movement				
2-8 weeks	Vestibular Reaction (slow & fast phases)				
3 months			Counting fingers	1" black cube against white surface at 2'	
6 months	Further development of the Optically Elicited Movement response. ↓		20/400	1/3" black cube against white surface at 2'	
1 year			20/200	20/240	
2 years			20/100	20/50	
3 years			20/50	20/50	15/30
4 years				20/30	20/30

sessed. In man, OKN is dependent upon the presence of cerebral function. While it has been stated frequently that OKN cannot be elicited until some time (3-6 months) after birth, when optic fixation is developed, the work of Gorman et al.<sup>8</sup> indicates that this response is present within the first few hours of life. Using a test pattern of alternating black and white lines which occupied almost the entire visual field of 100 infants 1½ hours to 5 days old, these workers found that 93 of them could perceive a pattern which, at the test distance used, corresponded to a Snellen acuity of 20/670. Reinecke and Cogan<sup>12</sup> have found that the Snellen visual acuity of a given subject is usually better than the acuity determined by the OKN response. Thus, when an OKN response is elicited by a test pattern of lines of a given width, the patient can see at least the Snellen equivalent of the lines, and usually better. In young children then, or others incapable of trustworthy subjective responses, the objective measurement of vision by means of the OKN response gives a reliable indication of a minimum visual acuity.

FIRST SIX MONTHS

As the infant's visual mechanism develops, other methods by which this vision can be assessed are available. *Optically elicited movement* is defined by Kestenbaum<sup>7</sup> as that movement of the eyes toward a peripherally appearing object. It is in part the function

of a peripheral retinal point but apparently is also dependent upon the presence of central vision of a higher acuity than the peripheral retina. Thus, at 4-6 weeks of age the child may respond to a peripherally presented flashlight or large object by turning the eyes toward the stimulus, as if to fixate upon it. Initially, these efforts may be quite transitory and considerable patience may be required before the examiner can determine whether or not they represent a *bona fide* response to the stimulus. A little later (5-6 weeks of age), some attempt to follow a large moving object may be observed. These brief following movements are initially jerky, or cogwheel, in nature and represent an attempt to keep the image of the moving object upon the macula. It is not until 4-6 months of age that the following movement becomes gliding, indicating the development of coordination of the central fixation and ocular motor pathways. Utilizing this response, one may examine the visual field of infants as young as 4 weeks, and gain some idea of gross defects such as a hemianopsia. If the visual field is intact, then the optically elicited movements will serve to demonstrate the state of ocular motility. As the visual mechanism develops, the optically elicited movements are more consistently elicited; smaller objects are perceived; following movements are more sustained and more coordinated. By 3-4 months of age, the normal infant will follow a 1 inch black cube against a white background at a distance of 2 feet; by 6 months

TABLE II

SIZE OF TEST OBJECTS AND APPROXIMATE SNELLEN ACUITY EQUIVALENTS (after Schwarting (9))	
Diameter of Test Object in millimeters	Approximate Snellen Equivalent
20.00 (disc)	Counting (less than 20/400) Fingers
1.50	20/400
0.75	20/200
0.35	20/100
0.15	20/50

of age, the infant will follow a 1/3 inch black cube at the same distance.

A variety of methods have been devised for ascertaining the visual acuity utilizing the optically elicited movement, or following, reflex. Schwarting<sup>9</sup> used steel wires of different widths on a metronome moving at a rate of 40 half-cycles per minute. The vision was tested at a distance of 1 meter in the dark with the wire moving across an illuminated field. The thinnest wire which elicited a smooth synchronous following movement for at least one-half cycle indicated the visual acuity. The diameter of the test objects and their approximate Snellen equivalents are listed in Table II and the average visual acuities for a group of normal children tested by Schwarting are given in Table I.

Evans<sup>13</sup> devised a simple test using black iron filings which are moved about on a white opaque tray by means of a magnet manipulated underneath the tray by the examiner. The iron filings are graded by size and the smallest filing which attracts the child's attention indicates the visual acuity. Evan's felt that many children as young as 3 months can give reliable responses to this test.

A less refined technique is that used by Friedman,<sup>14</sup> who uses it primarily to detect unequal vision in the two eyes, as in amblyopia ex anopsia.\* He places a few nonpareils, the tiny colored candies used to decorate cakes, in front of the child and observes how promptly and accurately the child picks them up. Then he alternately covers one of the child's eyes and notes any difference in performance. This simple test, dependent in part upon the coordination of hand and eye, can also be conducted at home by the mother and has the virtue of eliminating the distractions of the doctor's office.

OLDER CHILDREN (1-4 YEARS)

In addition to the methods described above, other techniques may be used in the toddler or ambulatory child. Worth<sup>15</sup> threw white marbles 1/2 to 1 1/2 inches in diameter to different corners of the room, using a

\* Amblyopia ex anopsia is a reduction of vision due to suppression.

slight spin to change the direction of the sphere. Then he observed the accuracy with which the child sought out and retrieved the marble, and thereby deduced a level of visual acuity. In practice, however, this method is not entirely satisfactory because it is rather time-consuming and demands cooperation on the part of the child. As a general rule, the farther one gets away from the child when testing vision, the less the child's interest is sustained. The use of other small objects — pins, beads, coins, toys, etc. — at a closer distance is usually more satisfactory in determining visual acuity, particularly when a comparison of the two eyes is desired. It is helpful to have the child's mother cover one eye while the other is tested since the child is generally more amenable to being handled by the parent than by the doctor.

Often a definite amblyopia can be detected simply by observing the child's response when the eye with better vision is covered. In such instances, when an object of interest is shown to the child, he will vigorously resist occlusion of the better eye but will not object to occlusion of the amblyopic eye. Similarly, Rychener<sup>16</sup> observes the child's response to the "pirate game" in which the child's eyes are alternately covered by a black patch and the ease with which he "explores" the *terra incognita* of the doctors office is noted. Parenthetically, it should be stated that great care must be taken to assure that the occluded eye is indeed occluded. Children are great "peekers" and an erroneous assumption may be made that good vision exists in what is actually an amblyopic eye simply because the better eye is not covered. Thus, when a child attempts to use the occluded eye by twisting or turning during the testing of the fellow eye one may suspect unequal vision in the two eyes. Similarly, if the child repeatedly guesses at the identity of the object with one eye but answers correctly and promptly with the other, unequal vision is quite probable.

When the child is old enough to name familiar objects, a picture test such as that devised by Allen<sup>4</sup> can be used. This test consists of a series of black pictures on individual white plastic cards. The pictures are so constructed that they correspond as closely as possible with the Snellen letter E used at 30 feet. The pictures are shown to the child close at hand so that he can recognize and name them. Then with one eye covered, the child is asked to name the pictures as the examiner backs away and presents them individually. The greatest distance at which the child recognizes the pictures is the numerator of the Snellen fraction, the denominator for which is 30. Thus, normal three-year-olds can recognize the pictures at 15 feet and have, therefore, a visual acuity of 15/30; four-year-olds normally can recognize them at 20 feet (visual acuity of 20/30). It is possible to obtain a fair estimate of the visual acuity of many children between the ages of two and three years.

Recently, Allen<sup>17</sup> has presented a near-vision test utilizing the same figures scaled down for shorter test

distances. One advantage of this test is its usefulness in testing monocular myopia to determine the presence or absence of amblyopia in the myopic eye.

Other test charts utilizing pictures<sup>18,19</sup> have been devised. In my hands, these are less satisfactory than the Allen test cards because the presence of many figures on the chart tends to distract the child and they are intended for use at a fixed test distance which reduces the facility with which the test can be given.

The "illiterate E" test represents the last step before the standard Snellen letter and number charts can be used and, for those children capable of performing it, is probably the most reliable. In this test, the letter E is presented to the child in any of four cardinal positions (up, down, right and left) and the child indicates the position of the letter by naming the direction in which the arms of the E point or by holding his hand in the comparable position. A variant of this test has been devised by Sjogren<sup>20</sup> using pictures of a hand with fingers extended, pointing in any of four positions. The test letters may be presented in different sizes on one chart at a fixed distance as in the standard Snellen charts or a single E may be used at various distances as in the Allen test figures. The age at which this test can be used varies considerably. There are five-year-olds who, although apparently normal in other respects, do not seem to understand the test at the first examination well enough for reliable visual acuity determinations. For these children, the picture test may be satisfactory and the mother can instruct the child in the "E game" with a homemade block-letter E to familiarize the child with it for use at a later time.

A final word should be said about strabismus since it frequently leads to amblyopia ex anopsia in the deviating eye. This form of impaired vision is remediable in most instances if detected in the early years of life. If amblyopia persists beyond the age of five or six years, the chance of significant improvement in vision is markedly lessened. If for no other reason than this, the assessment of vision in the pre-school child is of utmost importance. At times, it may be difficult to determine the visual acuity in a young child by any of the methods described above. If, however, the child will fixate a flashlight and it is evident from the position of the reflected images of the light source on the cornea that one eye is deviating, referral to the ophthalmologist is in order. If, on the other hand, the corneal light reflexes appear symmetrical *and no other abnormality is noted*, it is justifiable to wait until the child is a few months older at which time the visual acuity may be determined satisfactorily. It should be remembered that many cases of amblyopia

may be due to a refractive error in the *absence* of strabismus. For this reason, one should persist until a reliable assessment of the child's vision is made.

#### SUMMARY

In view of the great importance of early vision testing in the detection of ocular defects, particularly those which are thereby rendered remediable, a summary of techniques useful in assessing the vision of the pre-literate child has been presented. The physicians in general and pediatric practice can join with ophthalmologists in attacking this significant problem for, as W. H. Fink<sup>1</sup> has said, "The physician's responsibility for the child's eyes begins before birth."

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# Adenomyosis\*

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Adenomyosis of the uterus is defined as the benign invasion of the endometrium into the myometrium together with a diffuse overgrowth of the latter. The historical background of this entity dates back to 1860 when Rokitansky first described this histopathology. Confirmatory descriptions were made by other observers, including Von Recklinghausen in 1896. However, it remained for Cullen, in his classic monograph in 1908 entitled "Adenomyoma Uteri,"<sup>3</sup> to demonstrate the anatomical continuity between the endometrium and the ectopic foci of endometrial glands and stroma in the myometrium. In his series of 73 cases, Cullen proved, by serial sections, this communication in 56 cases. Further, this continuity was found in all those cases where the ectopic endometrium was limited to the inner third of the myometrium. Cullen described the characteristic symptom complex of excessive dysfunctional bleeding and progressively severe acquired dysmenorrhea with the physical finding of an enlarging, firm, tender uterus. The term *endometriosis interna* has also been applied to this entity, as has *adenomyoma* for the more circumscribed form. Endometriosis interna is a slightly misleading term because adenomyosis involves both endometrium and hyperplasia of the myometrium, whereas pelvic endometriosis involves only endometrium. Further, most observers feel that the endometrial implants of adenomyosis do not usually respond cyclically as do the ectopic areas of endometriosis.

Grossly, the process of adenomyosis may produce a slight to moderate diffuse uterine enlargement, more often involving the posterior myometrium. Novak states that "rarely is such a uterus larger than a large orange."<sup>11</sup> Benson and Sneed noted that adenomyosis alone rarely caused extreme uterine enlargement over 200 grams. On sectioning the uterus, the myometrium is diffusely thickened and may exhibit discrete small areas of old or recent hemorrhage scattered throughout or minute cystic spongy areas. The associated endometrium may vary from normal to thickened, polypoid tissue. Microscopically, the hemorrhagic or cystic myometrial areas are composed of fairly typical endometrial glands and stroma. According to Novak, these glands occasionally exhibit the usual cyclic response but more often respond only to the estrogenic stimulus developing proliferative or cystic hyperplasia pattern.<sup>10,11</sup> This variability of gland response accounts for the

differences in the gross appearance of the sectioned myometrium in this entity.

The origin of this ectopic endometrium was initially considered to be from embryologic rests such as Wolffian duct or Mullerian duct remnants. However, Cullen's monograph had led to the general acceptance that this arises from the endometrium lining the uterine cavity. Blain and Brines postulate a spontaneous generation of endometrial stromal or interstitial cells from undifferentiated myometrial tissue, which subsequently develops into epithelial cells with gland formation. Curtiss postulated lymphatic or vascular spread of endometrial cells to explain adenomyosis found mainly in the outer third of myometrium.<sup>12</sup> The proponents of Cullen's theory cannot establish the underlying cause for the benign endometrial invasion. They postulate that, since adenomyosis is an increased growth activity of normal tissues, and since estrogen is a precursor to such growth, there may be some underlying ovarian dysfunction as the initiating factor<sup>10,13</sup> but there is no definite experimental proof of this concept to be found in the literature.

As previously mentioned, the symptoms usually cited are menorrhagia and progressive dysmenorrhea, with premenstrual tension and pelvic pressure listed as ancillary symptoms. Intermenstrual bleeding is quite rare in the experience of most observers. However, Hunter et al, in a series of 110 cases of uncomplicated adenomyosis, listed intermenstrual bleeding in 42 cases, with excessive menses noted in 85 cases and dysmenorrhea in 61. Also, a tender uterus was noted in 60 cases. Emge stated that the symptoms of excessive menses and progressive dysmenorrhea were not diagnostic, and that the development of a uniformly firm, enlarging, tender uterus alone should lead to the proper diagnosis of adenomyosis. The association of endometrial hyperplasia with adenomyosis varied from negligible in Hunter's series to 40% in Novak's series,<sup>10</sup> with such intermediate values as 10% (Benson & Sneed), 11% (Israel & Wouters), and 25% (Henderson).

There is general agreement that the predominant symptoms of dysmenorrhea and excessive menses are due to impaired myometrial contractility secondary to the interposed endometrium and the increased vascularity of the hypertrophied myometrium, although Benson and Sneed and also Israel and Wouters, stated that one-third of their cases showed up as incidental and asymptomatic. Generally, adenomyosis is found most frequently in women in their fifth decade. Israel and Wouters say more are found in whites than in Negroes. Ten to 30% of all hysterectomy specimens are found to have adenomyosis.

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Although Emge states that he correctly diagnosed adenomyosis preoperatively in 64% of 147 "typical" cases, the general diagnostic acumen has been much lower, ranging from 4% (Israel & Wouters) to 9% (Benson & Sneed). And, indeed, in Emge's series 117 cases with other preoperative diagnoses were found to have adenomyosis, thus lowering his diagnosis rate to 36%.

With the generally low incidence of preoperative diagnosis, ancillary diagnostic procedures would seem to be indicated. Such procedures as endometrial biopsy are inadequate, and myometrial biopsy is generally condemned, presumably because of the hazards of hemorrhage or perforation. Israel and Wouters state that 6 of their 307 cases were diagnosed by preliminary uterine curettage, but no other report listed this as efficacious in diagnosis. Uterography utilizing water soluble contrast media such as Skiodan acacia has established roentgenographic diagnosis in 38 of 150 cases (26%) of adenomyosis, as reported by Marshak and Goldberger. The typical finding described is short (2-4mm.) tubular passages perpendicular to the uterine cavity ending in sacular formations.<sup>5,9</sup> Other suggestive findings are marked irregularity of the uterine cavity border, or a marked filling defect in the cavity. They state that the findings can be differentiated from endometrial hyperplasia which shows an indistinct uterine cavity with multiple smooth filling defects, and any tubular spicules present parallel the myometrium and are not connected. There have been no subsequent reports corroborating the success of this procedure in the literature. Therapy of adenomyosis consists of hysterectomy, with radiation only when surgery is contraindicated.

My retrospective survey of adenomyosis in the New York Upstate Medical Center covers the period from January 1, 1955 to December 31, 1959 and includes all cases at hospitals associated with the Medical Center. The diagnosis and surgery was accomplished by resident physicians and private physicians, both gynecologists and general surgeons. There were a total of 159 cases in this survey. Discussion with the pathologists who determined the microscopic diagnosis showed the criteria to consist of the presence of endometrial glands and/or stroma in a hypertrophic myometrium, separated from the endometrial-myometrial junction by at least one low-power field. The age range was from 28 to 73, with 85 cases or 60% occurring in the 40-49 year age group. Most, 100/159 or 63%, occurred in women with one to four pregnancies and 10% in nulligravidas. Nineteen of these cases or 12% were diagnosed preoperatively, 5 by preliminary D & C.

The symptomatology listed in the various protocols included the following:

Excessive menses in 78 cases with secondary anemia cited in 10 of these. Menstrual irregularity was noted in 39 cases, with postmenopausal bleeding cited in 12 cases. Pain was an important feature of this series,

occurring in 72 cases. However, dysmenorrhea was specified in only 24 patients, and premenstrual tension listed in 5 cases. The factor of uterine enlargement was mentioned in 56 cases. However, since myomata were diagnosed in 74 cases, this finding of uterine enlargement lost much of its significance. In fact, in only 20 out of the 85 cases or 24% of adenomyosis without associated myomata was this noted. Other associated diagnoses were endometriosis in 29 cases, and endometrial hyperplasia in 69 or 44%. In a supplementary survey of cases of myomata only, 10% had endometrial hyperplasia. Also, in the cases where adenomyosis was the sole significant diagnosis, 70% had associated endometrial hyperplasia.

This last associated finding seemed significant compared to other reports, so it was decided to amplify the survey. Accordingly, the gynecological pathology reports at the Upstate Medical Center were reviewed over the period from 1945-1954. A total of 500 cases of adenomyosis were collected with associated endometrial hyperplasia found in 282 cases or 56%. In those cases where adenomyosis was the only significant uterine pathology (96 cases), there was 79% associated endometrial hyperplasia.

This survey confirms generally the impression that adenomyosis is found most commonly in parous women in the fifth decade of life, with a history of excessive menses, dysmenorrhea, and uterine enlargement, although the latter two findings were not as prominent in this series as in previous papers on the subject. However, the incidence of associated endometrial hyperplasia seems sufficient to entertain the preoperative diagnosis of adenomyosis when endometrial hyperplasia is present in a patient fulfilling the other criteria. Further, in patients where the symptomatology and physical findings are equivocal, uterography is suggested as a possible method of confirming the diagnosis of adenomyosis.

#### SUMMARY

1. An initial survey of 159 cases of adenomyosis revealed that endometrial hyperplasia was an associated finding in 44% of the cases.

2. In those cases where adenomyosis was the only diagnosis, 70% had associated endometrial hyperplasia.

3.a A supplementary survey of 500 cases of adenomyosis was conducted and endometrial hyperplasia was an associated diagnosis in 56% of these cases.

3.b Where adenomyosis was the only significant pathology, 79% had associated endometrial hyperplasia.

The incidence of endometrial hyperplasia in conjunction with adenomyosis in this series indicates this finding should be helpful in establishing the preoperative diagnosis of adenomyosis by endometrial biopsy or preliminary curettage.

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## THE DIAGNOSIS AND TREATMENT OF NASOPHARYNGEAL CONDITIONS — *Continued from Page 29*

their origin in the foetal notochord, have been reported in the literature. These are of slow malignancy but tend to recur after operation and the prognosis is unfavorable. Occasionally pituitary tumors, such as the chromophobe adenoma, may invade the floor of the sella turcica and present in the nasopharynx.

Often the first sign of nasopharyngeal malignancy is metastatic cervical adenopathy. In all cases of unexplained nodes nasopharyngeal neoplasm should be suspected. Unexplained cranial nerve involvement with or without cervical adenopathy, should suggest the possibility of nasopharyngeal malignancy.

Intensive irradiation generally is the only form of therapy to be considered. While certain of these tumors, especially the lymphoid type, are themselves radio-sensitive, the metastases to the cervical glands are very resistant, so that treatment is apt to be little more than palliative. If nodes exist before therapy, radical neck dissection is indicated. While the undifferentiated lymphoepithelioma may respond to X-ray therapy, generally the prognosis is poor.

The recent advances in chemo-therapy for malignancy may indicate a more effective way of managing these conditions. A case of the author's, a rhabdomyosarcoma in a boy of seven, with metastases to the skull, the pelvis, and the lungs, had shown what seemed to be encouraging response to this method of therapy for a period of several months, before finally death ensued. At present, however, this is only something to hope for.

The applied anatomy of the nasopharynx has been briefly reviewed (together with the technique of examination). The significance of adenoids and the indications for operation, as well as the technique has been discussed. Follow-up studies of operated cases indicate the importance of visual control. Other conditions of clinical importance include the hypertrophy of the posterior tips of the inferior turbinates, choanal polypi, posterior choanal atresia, cysts and foreign bodies. Juvenile basal fibromata often present problems in diagnosis and management. Malignant tumors in the nasopharynx often may be overlooked. The prognosis is generally poor.

# A Plea For Pyelo-Plastic Procedures

EARLE M. DAVIS, M.D.\*

The plastic procedures referred to in the title are those which have been performed on the uretero-pelvic junction obstructions seen frequently in all age groups and which give rise to progressive hydronephrosis and eventual loss of renal function. The first such plastic procedure was described in 1886 by Trendelenburg, and since that time many modifications and new procedures have been employed. In 1937 Foley<sup>1</sup> listed three principles which must be followed in repairing any uretero-pelvic obstruction, from whatever cause. The test of time has proven these concepts: 1.) low insertion of the ureter into the pelvis, 2.) smooth, funnel-shaped junction, and 3.) no shortening of the suture line. If this site of obstructive pathology is kept in mind in the management of all chronic or recurrent urinary tract infections, and if the plastic operative procedures are utilized properly, secondary cardio-vascular complications and some nephrectomies may be avoided.

One of the basic principles which guides urologic surgery, and which should be kept in mind during the medical management of any urinary tract disease, is the attempt to preserve as much functioning renal tissue as possible. The early recognition of congenital conditions will lead to their early correction and prevention of progressively destructive lesions. However, even if the condition is not discovered until the middle years of life, as is often the case, and there has already occurred extensive damage to the kidney, there is no reason for perfunctory nephrectomy. The argument that it is more harmful to retain a poorly functioning kidney, which will lead eventually to severe cardio-vascular complications, is far from substantiated and should not be used as a reason for performing a nephrectomy instead of a more difficult plastic repair, with its more involved post-operative care.

In the first place, it is difficult to prove to what extent renal function has been lost. The increasing use of radio-active hippuran excretion measurement is demonstrating that the older method of judging renal function by the appearance of radio-opaque dye, both in time and concentration, on the intravenous pyelogram, has been misleading in many instances. In the second place, there is no proven method of predicting how much function will return, once the obstructive lesion has been corrected. Certainly the method of bilateral renal catheterization and measurement of PSP excretion is often misleading. Unfortunately the

forecasting of the amount of renal function which should return following proper surgery is a matter of judgment, and this is based on past experience and the condition of the patient in general and of the kidney in particular. Past experience has shown that it is worth salvaging these damaged kidneys, and the ever increasing armamentarium of the pharmacologist gives even more hope for the future.

In any case where there has been persistent or recurrent, unexplained pyuria, an intravenous pyelogram should be done to rule out upper urinary tract obstruction. If an apparently non-functioning kidney is found, or a hydronephrotic kidney with poor concentration of dye over a longer than usual period, retrograde pyelograms should be made. In our practice, we have found it of much greater value to perform the cystoscopic procedure under local anesthesia and thus have the full cooperation of the patient. The sometimes hugely dilated renal elements may be safely distended with dye without the fear of overdistention and the consequent backflow which may give rise to bothersome acute reactions. If the patient is able to cooperate, special views of the obstructed kidney are often possible which will more clearly demonstrate the actual point of obstruction and its nature.

In preparing the patient for plastic surgery, the active pyelonephritis which is almost invariably present should be under as much control as possible with chemotherapeutic or antibiotic agents. Patients should be warned that a good liquid intake post-operatively will be necessary, and that renal function and infection must be checked frequently in the future, to assure success in the procedure. They should also be warned that post-operatively, for a few days at least, a splinting catheter will emerge from their incision and they may drain urine for a short time following its removal. A completely cooperative patient is essential to the happy outcome of pyeloplastic procedures, and if this cooperation cannot be expected, it is probably wiser to plan the quicker, and possibly less complicated, nephrectomy.

The choice of the particular procedure to be used, and it is an extremely wide choice, depends upon the experience of the surgeon and the conditions that exist in the particular case. As in most things on earth, the simpler the procedure, the better the results, as long as the basic principles as laid down by Dr. Foley are followed. There has always been great discussion among the urological surgeons as to the wisdom of using or avoiding ureteral splinting catheters and by-passing drainage via nephrostomy or pyelostomy

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FIGURE I. Intravenous pyelogram, pre-operatively, showing practically no function on the right side, even after 30 minutes. There was normal function of the left side with good filling of a normally shaped renal pelvis and uretero-pelvic junction.



FIGURE II. Retrograde pyelogram, pre-operatively, showing moderately distended right renal pelvis, with high insertion of the ureter, giving a deformed uretero-pelvic junction. The calyceal systems show only moderate changes, consistent with intermittent obstruction over many years.

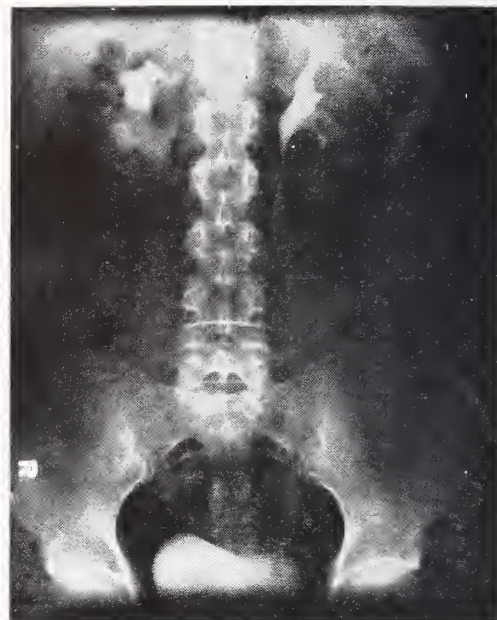


FIGURE III. Intravenous pyelogram, 10 weeks following right pyeloplasty, showing prompt excretion of normally concentrated dye and proper emptying of a normally shaped renal pelvis.

tubes. In our practice, we have found it most useful, and sometimes reassuring, to have a large calibre, perforated polyethylene splint passing up through the repaired uretero-pelvic junction, out through a small, highly placed pyelostomy, and emerge from the skin incision along with the penrose drain. This tubing is left open and draining for twenty-four hours, closed but in place for another forty-eight hours, and removed on the third or fourth post-operative day. We believe that most of the reasons given for not using splints are resolved by this type of tubing. Polyethylene causes little or no tissue reaction, the multiple perforations prevent plugging with subsequent pelvic distention, high fluid output and brief time of intubation avoid incrustation with possible subsequent stone formation, and the early and easy removal avoids secondary infection. Because we try for a water-tight closure, using 4-0 chromic sutures with mucosa apposition, the pyelostomy and tube can be reassuring on the second or third post-operative day when edema could conceivably be obstructing the ureteral end of the repair.

In the last three years we have performed seven pyeloplasties, the youngest patient was a fourteen year old girl and the oldest a seventy-four year old man with an hypoplastic, non-functioning contralateral kidney. All cases, no matter what age, gave past histories of recurrent backache, recurrent lower tract complaints of varying degrees, and showed variable but persistent pyuria. Follow-up studies have shown only slight improvement of the x-ray appearance by intravenous pyelogram, but every case has been free of backache and has had no recurrence of lower urinary tract complaints. Microscopic pyuria has persisted in all cases

when chemotherapy was stopped, but many specimens show only rare cells, and in two cases colony counts have been less than 1,000/cc. Colony counts have not been done on the earlier cases as yet, but will be done should microscopic pyuria be found in their future examinations.

An illustrative case is that of a forty-eight year old housewife who had been enduring stoically intermittent right costo-vertebral angle and upper quadrant pain, associated with vomiting at times, fever, dysuria, and pyuria. Her episodes had been less frequent in recent years, but on the most recent one, she consulted a physician. When he discovered the pyuria, an intravenous pyelogram was done and a "non-functioning" right kidney was seen. Retrograde pyelograms clearly revealed the uretero-pelvic junction obstruction and culture of the right pelvis urine gave no growth of bacteria. The diagnosis of intermittent hydronephrosis with chronic pyelonephritis was made. A revision of the uretero-pelvic junction was done following the excision of an excessive amount of peri-pelvic and peri-ureteral fibrous tissue. Because of the extra handling of the tissue necessitated by this procedure, the polyethylene splint was left in place for eight days. There was no urinary drainage following its removal, and the eleventh post-operative day saw the patient discharged with a dry wound. Eight weeks post-operatively, a clean voided specimen of urine showed only a rare white blood cell and the patient was comfortably carrying on activities only somewhat limited.

In recapitulation, it should be emphasized that anomalous conditions in the region of the uretero-pelvic junction may give rise to symptomatic difficulties later

in life than childhood, and that the earlier they are found and corrected, the happier the results in saving renal tissue. Any persistent or recurrent urinary tract infection calls for intravenous pyelogram, and this in turn may demand careful retrograde studies. Proper pre-operative evaluation will preclude perfunctory nephrectomy, while the appropriate pyeloplastic procedure along with thorough post-operative care will salvage many useful kidneys, even though "perfect

function" may not result. Just how much eventual secondary complications in other systems may result from the retaining of these imperfectly functioning renal elements will have to be determined by long and careful observation. In the meantime, however, nothing functioning has been lost and there is opportunity for regaining already lost function.

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## Tetanus—A Case Study

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Ten deaths due to tetanus have been reported in this state during the past ten years. Tetanus toxoid is so effective prophylactically that without knowing the details of any of the cases, it is probably safe to say that both the disease and the deaths were unnecessary. However, cases of tetanus do occur, with problems of diagnosis and treatment. In the case to be presented, the problem of clinical interest revolved around the difficulty of making an early diagnosis, and then the question of whether treatment with horse serum and antibiotics was necessary or desirable. Statistics are available indicating the survival rate of tetanus cases not receiving such specific treatment.<sup>1</sup> No figures could be found indicating how many, if any, fatalities could be attributed to such treatment. Horse serum is a notorious drug to produce anaphylactic reactions; and overzealous use of depressant drugs used to combat strangulating spasms could conceivably prove fatal. It may be that some mild cases of tetanus are not diagnosed, not treated, and not reported. Possibly this is because it is difficult to confirm the diagnosis of mild tetanus, and possibly because it is even more difficult to justify the withholding of specific treatment once the diagnosis is made.

According to the Mayo Clinic experience,<sup>2</sup> the mortality varies with the incubation period, the rapidity with which symptoms develop, the severity of the disease and with age. A short incubation period is accompanied by a high mortality. When the incubation period was nine days or less, the mortality rate exceeded 60%. When the interval of time from the onset of symptoms to the maximal severity of the disease was four days or less, the mortality rate was about 70%. In patients classified as having mild, lo-

calized disease, there was 100% recovery with treatment. In patients over forty years of age, the mortality exceeded 50%. This paper does not indicate how many of the mild cases, if any, actually had a rapid development or a short incubation period. No statistics could be found correlating the mortality rate with the day of the disease on which treatment was started.

An interesting study was carried out<sup>1</sup> in which two similar groups of patients with tetanus were rendered identical treatment, with the exception that one group received large doses of tetanus antitoxin and the other group received none. The death rate was 49% in patients receiving serum, and 76% in the patients not receiving serum. It seems remarkable that 24% of unselected cases could survive without serum. Presumably this constitutes the percentage of cases which are mild and localized throughout their natural course. The question is not discussed regarding the possibility of recognizing these favorable cases with sufficient accuracy as to justify treatment without serum. The mechanism of death in tetanus frequently has to do with generalized convulsions and strangulation, bronchial aspiration and pneumonia, or inanition from inability to swallow. It would appear that cases not so involved are capable of spontaneous recovery. Probably a diagnosis of mild, localized disease can only be made in retrospect.

It is highly questionable if valid appraisal can be made of a case in which specific treatment would not be indicated. On the contrary, the indications for treatment have been unequivocally stated by Stafford:<sup>3</sup> "Recognition of patients whose situation demands antitetanic therapy: 1. Any patient with or without a history of recent injury who complains of inability to open his mouth properly, or who presents localized muscle spasm, or who is having generalized tonic

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spasm of skeletal muscles. 2. Antitetanus therapy may be withheld if a positive diagnosis can be made of any of the following conditions which may simulate tetanus: masseter spasm due to dental infection or local cellulitis; acute arthritis of the temporo-mandibular joint; tonic spasm caused by brain stem disease or injury; tetany caused by rickets or chemical imbalance, as in severe vomiting; trismus or tonic spasm resulting from the use of prochlorperazine or related drugs."

In the case under discussion the administration of antitoxin was electively deferred, mostly because of the apparent benign course of the disease. We were somewhat influenced by the fact that the patient was an asthmatic with a history of many drug reactions. She had never been inoculated with tetanus toxoid; but there was no known sensitivity to horse dander or horse serum. Nevertheless, in view of the strong allergy history, it appeared desirable to avoid the use of serum and antibiotics if possible.

It was anticipated that the course of events might make intensive treatment mandatory; and the possibility that the patient would be positive to a skin test of horse serum was considered. Skudder<sup>4,5</sup> states that over half of all individuals in New York City are sensitive to horse serum. This figure is contrary to all other reported experience, and probably represents a gross error in technique or in the interpretation of the tests. Nevertheless, at times it is urgently necessary to decide what course to follow when a patient is, in fact, allergic to horse serum. The alternative procedures to the use of horse serum are the use of bovine tetanus antitoxin, or human hyperimmune serum. An effort can be made at rapid active immunization with intradermal tetanus toxoid; or reliance can be placed on surgical excision, when feasible, of the presumed site of infection, plus the use of antibiotics. In regard to surgery, the problem of what to do with a well healed wound can involve some difficult decisions. It has been advised<sup>6</sup> that if the site of infection involves a digit, amputation should be carried out, even if the wound is well healed.

There is an established procedure for administering horse serum to a sensitive patient, starting with a minute dose, and gradually increasing. Recently doubt has been cast on the efficacy of horse serum when given under such circumstances. Although the problem has not been entirely resolved, there is reason to believe that the antitoxin may be de-activated by combining with its specific antibody, and not be available to neutralize the tetanus toxin. Spaeth<sup>7</sup> states that "... TAT containing a foreign serum may be given but once in a lifetime (because of the) accelerated loss of antibody which occurs in the presence of acquired hypersensitivity."

#### A CASE REPORT

A sixty one year old female suffered a slight laceration

of her finger while preparing a meal. She had no fresh garden vegetables in the house, and had been doing no out-door work. It was painted with an antiseptic and healed uneventfully. Forty eight hours later she complained of difficulty in swallowing her supper. Twenty-four hours later she was unable to swallow meat. The next day she was examined for the first time, with no unusual findings. A day later she had difficulty swallowing liquids, and another examination revealed nothing significant. At this time a barium swallow under fluoroscopy appeared normal. The next morning she had difficulty opening her mouth, and was hospitalized with a question of tetanus. This was six days after her injury and the fourth day of symptoms. A consultation was held, and the opinion was that tetanus was unlikely forty eight hours after an injury; and that the patient probably was developing a local infection that produced dysphagia and trismus.

The next day her condition was unchanged. She felt good and had no discomfort. No type of stimulation could elicit reflex muscular spasms. Finally a vigorous attempt was made to examine her pharynx. This provoked a severe spasm of the muscles of her jaw and throat. She had difficulty breathing because of laryngospasm and mucus in her throat which she could not swallow. She became cyanotic and appeared in great distress. An airway was forced between her teeth, and she was suctioned out. In an hour she felt comfortable again, and was sitting up talking pleasantly. She stated that the experience had frightened her but had not been painful. Another consultation was held and it was decided to defer treatment. The disease was apparently not progressing. It was felt that this was a case of mild, localized tetanus, and that supportive treatment should be adequate. The thought was expressed that the hazard of treatment with horse serum and antibiotics might prove more dangerous than the disease.

On the ninth day of symptoms another mild effort was made to examine her throat. It was evident that she was not going to tolerate the procedure, and it was discontinued without disturbing her. Several hours later the patient suddenly had a spontaneous convulsion. The muscles of her jaws and neck went into massive, intense spasm. Within a minute or two there was generalized muscular involvement. The neck, back and extremities were in straight, rigid, extension. It was evident that she had difficulty ventilating, and she appeared to be straining to breathe. Her eyes moved from one to the other of the attendants around her; but she was otherwise immobile. She rapidly became increasingly cyanotic and then slumped unconscious.

Oxygen was immediately administered. With unconsciousness the patient appeared to relax enough to permit effective ventilation to occur. She was given 125 mg. of Sodium Luminal® by injection (injectable meprobamate was not available.) A tracheotomy was carried out using local novocaine anesthesia.

In the meantime, after doing skin tests which were negative, she was given a million units of penicillin and 100,000 units of tetanus antitoxin. She was maintained on 200 mg. of Solu-Cortef® daily to guard against reactions from these medications. During the seizure the patient's pulse was strong and regular. Peripheral tendon reflexes or a Babinski reflex could not be obtained. Previously, and for weeks afterwards, the tendon reflexes were hyperactive, and a sustained ankle clonus was present. Unfortunately it was not noted on what day of illness these neurological changes first occurred.

Following tracheotomy the patient's course was uneventful. She was comfortable and had no further seizures. No further efforts were made to examine her throat; but it was impossible otherwise to elicit any reflex spasms. No precautions were taken to avoid disturbing the patient. Her only complaints were inability to open her mouth, or to move her neck, or to swallow. Her neck ached mildly posteriorly. Actually, since she did not drool, she must have been able to swallow sputum. In two weeks she was able to take fluids by mouth. The spasm of the neck and jaw muscles subsided slowly, and disappeared in about six weeks.

#### COMMENT

A case of tetanus is presented in a sixty one year old female, an asthmatic with a history of many drug reactions. There was a delay in arriving at a diagnosis because the symptoms were mild, localized, and not progressive. Spasm of the jaws, elicited by manipulation of the throat, is presumedly a diagnostic feature of tetanus; so that a diagnosis should have been made at least at the time that this episode occurred. An unsuccessful, and almost disastrous, attempt was made to treat her supportively without horse serum or antibiotics, even after a diagnosis of tetanus was made. At the time of this decision, nine days of mild symptoms had elapsed, and it was not anticipated that the disease would suddenly become intensified. When a generalized, tonic convulsion occurred, and almost proved fatal from strangulation, maximum treatment was immediately instituted with tetanus antitoxin, penicillin, corticosteroids, sedation, and a tracheotomy. In retrospect it is not possible to evaluate the effect,

if any, of the serum or the antibiotic. The single generalized convulsion subsided spontaneously. The fact that there were no recurrences cannot be attributed to these medications, since it is known that no medication has any effect on toxin which is already fixed to central nervous system tissues.

A review of the recent literature and current textbooks did not disclose a good discussion of the dynamics of tetanus antitoxin. The reason for the effectiveness of a small prophylactic dose of antitoxin is obscure. The more pressing question regarding the efficacy of antitoxin with repeat use, seems to be unanswered at the present time.

#### SUMMARY

Tetanus is not a common disease, and it may be difficult to diagnose. In the absence of a typical clinical picture, and preferably before the disease progresses to this point, treatment should be started in the presence of certain suggestible findings. The fact that active treatment is such a formidable undertaking probably accounts for some early delay. Although about one fourth of tetanus cases are capable of recovery with supportive treatment, the course and severity of an individual case can be determined only in retrospect. It may be a dangerous mistake to attempt to identify the type of case which does require specific treatment. Once a diagnosis of tetanus is made, it would appear to be mandatory to institute immediate optimum treatment.

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# Case Report: Regression Of Metastatic Osteogenic Sarcoma On Chemotherapy

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In April of 1960, a sixteen-year-old boy was admitted to the hospital because of pain in the region of his right knee. He had no other complaints, and in all other respects he was physically in excellent condition. He had had no recent illnesses, nor was there any history of recent injury. He had played football in his high school the previous season, but received no particular injury to this area. His past history had been essentially negative, except for some of the usual childhood diseases. Family history revealed the mother and father to be living and well. There was no familial history of tuberculosis, malignancy, or diabetes. Systemic review was entirely negative. Physical examination revealed an extremely well-developed sixteen-year-old boy who was not apparently ill. He was 6' 4" tall, and weighed 235 pounds. Physical examination was entirely negative; the heart was entirely normal; the lungs were clear. Extremities were all negative, including the right lower extremity. The involved area revealed no tenderness, no discoloration, nor was there any swelling. A blood count was taken which revealed a hemoglobin of 15.4 grams; red blood count 5,400,000; white blood count 10,100; 58 polys, 41 lymphocytes, and 1 monocyte. The sedimentation rate was 16. X-rays of the chest were entirely negative. However, x-rays of the right lower femur revealed a moderately large bony defect. This was, subsequently, surgically explored and biopsied, and the pathologist's report revealed osteogenic sarcoma, (Figure 1 and Figure 2). On April 26, the right leg was disarticulated at the hip, and the lymph nodes of the right inguinal region were dissected out at the same time. The pathologist again reported osteogenic sarcoma of the bone, and no evidence of malignancy was found in any of the lymph nodes. The patient was subsequently discharged.

In late December of 1960, the boy was once again readmitted with the history of having had a cough for about one month. At the time of admission, he was raising bloody sputum, and was having moderate difficulty in breathing. X-rays were taken which revealed extensive metastases to both lungs, with a complete collapse of the left lung, and fluid was present in the left pleural cavity. There had been no weight loss, and the patient had felt well up until this point. The patient was treated surgically for this condition with evacuation of the bloody fluid from the left chest, and re-expansion

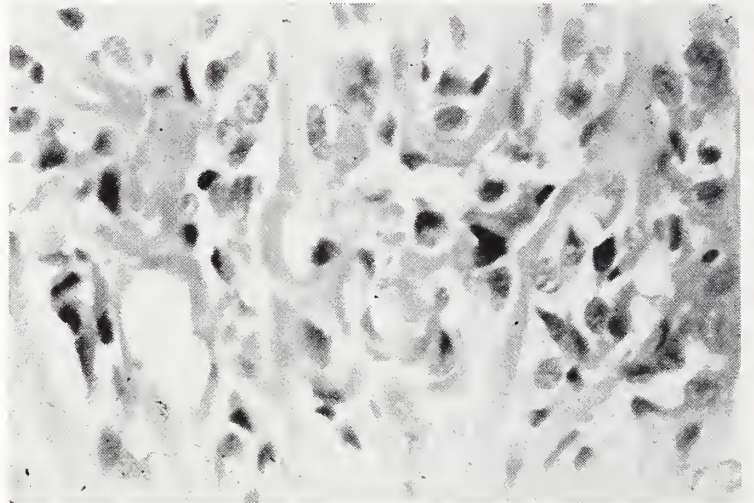


FIGURE 1

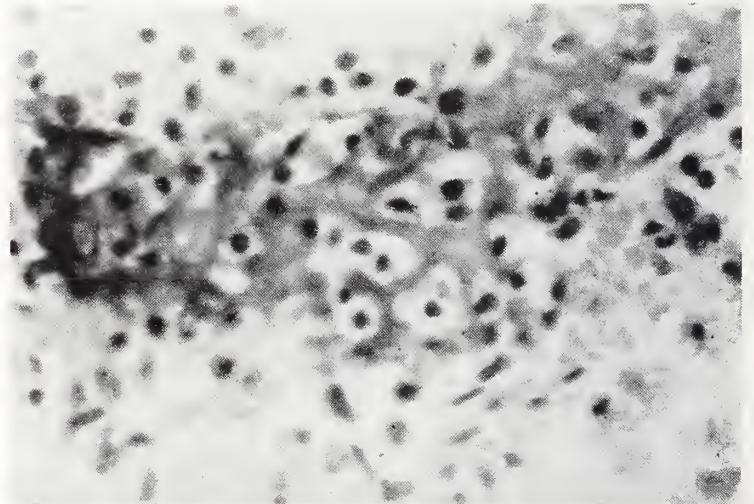


FIGURE 2

of the left lung. On January 7, 1961, the patient was seen for the first time by the chemotherapy service and was started on triple therapy. This regime involved a twenty-five day course of treatment, which included the daily oral administration of Chlorambucil, 10 mg., and Methotrexate, 5 mg., both given by mouth. On the third, twelfth, and twenty-first day of this regime, the patient was started on Actinomycin-D, 0.5 mg. daily intravenously, for five days. This was given into the tubing of a running intravenous solution of 5 per cent dextrose in water. On the eighth day of treatment, the day following the first course of Actinomycin-D, chest x-ray revealed essentially no change in the status of the pulmonary metastases, and Figure 3 dem-

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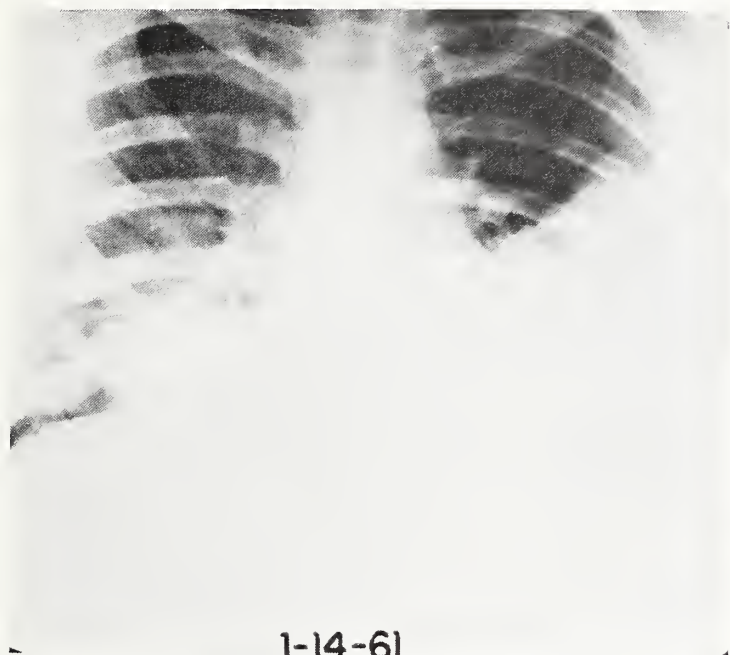


FIGURE 3

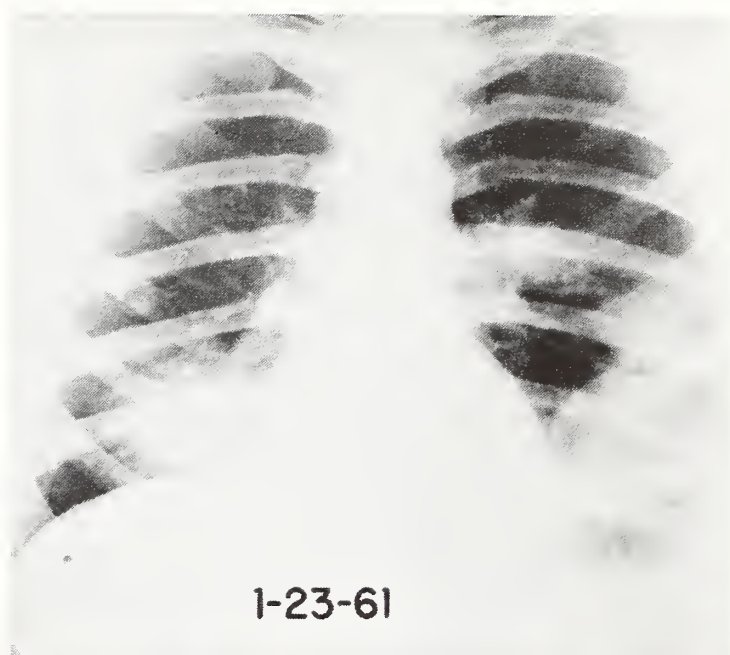


FIGURE 4

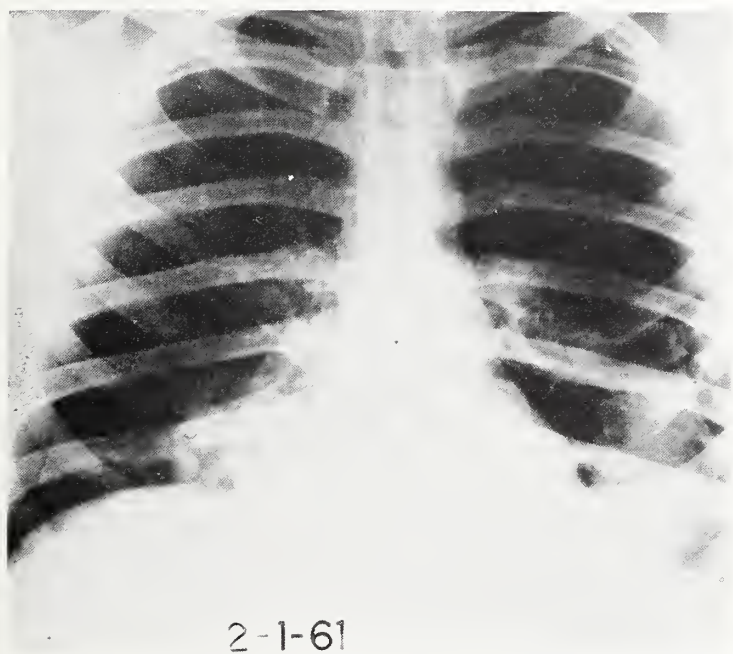


FIGURE 5

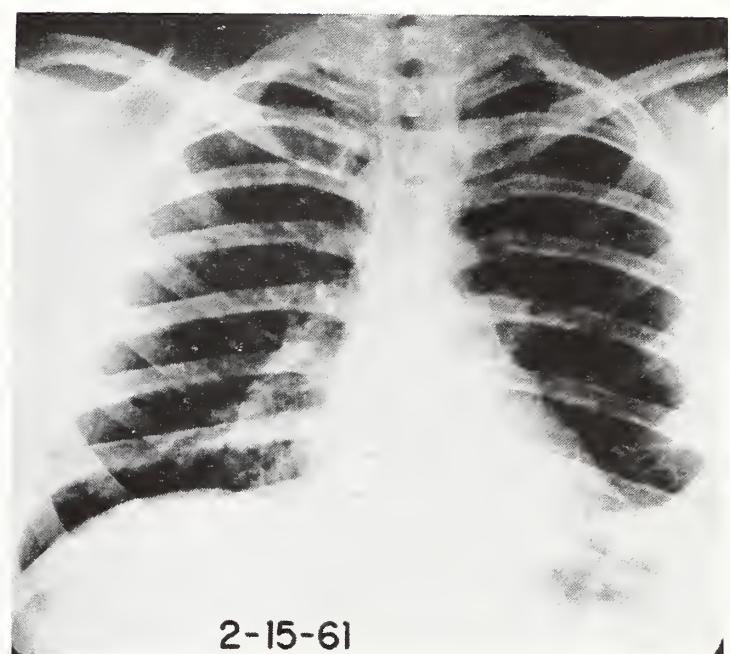


FIGURE 6

onstrates the basic picture of this patient's metastatic disease. A repeat chest x-ray on the seventeenth day of treatment, the day following the second course of Actinomycin-D, revealed, in addition to the reappearance of some pneumothorax on the left, a distinct and rather startling improvement in the picture of the pulmonary metastases, (Figure 4). On February 1, at the termination of the third course of Actinomycin-D, a chest x-ray revealed still further and more marked clearing of the pulmonary metastases, (Figure 5). On the 4th of February, catheters were inserted into the chest bilaterally for the relief of the mild pneumothorax which was seen on the last x-ray film, and in a day or so the pneumothorax had disappeared. On February 15, two weeks after the termination of the twenty-five day course of triple therapy, another chest x-ray was taken which, com-

paratively speaking, revealed only minimal evidence of pulmonary metastases, (Figure 6).

During treatment, no signs of toxicity developed, other than occasional vomiting following the administration of Actinomycin-D. On January 30, 1961, the blood picture revealed a hemoglobin of 12.8 grams; red blood count 4,500,000; white blood count 5,900; and platelets 128,000, the latter being the only sign of mild bone marrow depression. Throughout this course of treatment the patient was hospitalized, but was completely ambulatory on crutches, felt well, and had no complaints.

From the time of completion of the first twenty-five day course of triple therapy, the patient was given repeated seven-day courses of triple therapy with the Actinomycin-D being given on the last five of the

seven days. During this time he was an out-patient, and treatment was repeated every two weeks for two months. In May, the interval was increased to three weeks. Up to this point, repeated chest x-rays were unchanged and very satisfactory. The blood picture had been watched very closely, and there were no signs whatsoever of bone marrow depression. By the month of June, however, the chest x-ray showed some signs of increasing size of the remaining small metastases, and on June 3, 1961, the hemoglobin was 9.6 grams (62%); red blood count 3,100,000; white blood count 7,500; 75 polys, 22 lymphs, and 3 monocytes. The platelet count was 197,000, and the sedimentation rate was 29. In spite of the change in the blood picture, it was felt that triple therapy should continue because of the worsening of the chest x-ray, and the patient was given what actually turned out to be the last full course of triple therapy from the third to the ninth of June. On June 24, the hemoglobin was 7.4 grams (47%); red blood count 2,600,000; white blood count 6,500; with 81 polys, 16 lymphs, 2 monocytes, and 1 basophil, with a platelet count of 296,000. For the next month, he continued to be handled as an out-patient, and received several blood transfusions and no further chemotherapy, but by the end of July, his chest x-rays and blood picture, in spite of repeated blood transfusions for marked anemia, were becoming worse and the patient had to be readmitted. Stool examination subsequently revealed a 4 plus positive guaiac test. During the next few terminal weeks, and while we were supporting him with repeated blood transfusions, etc., the patient was given a short course of Chlorambucil and Methotrexate in an effort to narrow down which of the drugs might have been responsible for the tumor regression previously seen. However, the chest x-ray became steadily worse, and the anemia more pronounced. By mid-August, the hemoglobin was 7.8 grams; red blood count 2,900,000; white blood count 5,600; 94 polys, 5 lymphs, and 1 basophil. The hematocrit was 26 per cent; platelets were 101,000; prothrombin time 20/15; and sedimentation

rate was 81 mm. in one hour. Blood chemistries and electrolytes were all negative except for the alkaline phosphatase which was 5.1 Bodansky units, (normal 0 to 4); blood calcium 7.5 mg.%, (normal 8.5 to 11); and blood phosphorus 3.7 mg.%, (normal 2.5 to 3.5). The twenty-four hour urine calcium was 63 mg. per twenty-four hour period. (Normal 50 to 300 mg. per twenty-four hours). The significance of some of these slightly altered blood chemistries is not known. The Chlorambucil and Methotrexate had no apparent effect on the metastases as seen on repeated chest x-rays, and this was discontinued. The patient continued to go downhill, developed bilateral hydrothorax with increasing enlargement of pulmonary metastases and progressive worsening of his anemia. He subsequently expired in early September, nine months after onset of chemotherapy.

Significant postmortem findings were multiple metastatic sarcomatous lesions in both lungs, liver, spleen, and lymph nodes. There were bilateral pleural adhesions with a right hydrothorax. The right pulmonary veins were partially obstructed by extrinsic tumor. The gastrointestinal tract was completely negative, and there was no evidence of any areas from which bleeding might have come. The bone marrow showed marked hypoplasia with signs of early regeneration.

#### SUMMARY

This paper demonstrates what, to our knowledge, is the first example of tumor regression in a case of pulmonary metastases secondary to osteogenic sarcoma. This regression apparently occurred as the result of administration of three different chemotherapeutic agents which are known to have anti-cancer properties; namely, Chlorambucil, Methotrexate, and Actinomycin-D. The patient's life may have been prolonged by nine months, and during most of this time he was ambulatory, happy, and at home with his family.

82 Elm Street, Waterville, Maine



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### Healthy Added Years\*

EDWARD L. BORTZ, M.D.\*\*

The rapidly increasing number of older citizens, the majority of them in good health, since the turn of the century has been the result of the increasing efficiency of scientific medicine. With the use of only recently developed high-precision apparatus and techniques for study of the various diseases that destroy human health, more effective methods for treatment of these disorders have been elaborated. The result is a rapid decline in death rate with a corresponding extension of the life span of American citizens. This is all to the good. Now, the majority of infants may count on living a greater number of years than at any time in the history of civilization.

Since, until the last decade the elderly individual was much in the minority and could, relatively easily, be comfortably cared-for within the home, particularly on the farm, no problems arose.

The situation for families and communities has changed rapidly with the flow of large numbers of the rural population into the cities and towns. The presence of large numbers of healthy men and women beyond the 65-year-milestone, with limited facilities at home and at work has brought about a crisis of major dimension. Added to this, the continued march of scientific progress with the promise of greater control of those maladies which destroy life in the later decades, a further addition to the life span can, with confidence, be predicted. The four major maladies of the later years are diseases of the heart and circulatory system, cancer, arthritis and, finally, nervous and mental disturbances. The magic of science is now concentrating its big guns on these four principal diseases. Already a sufficient amount of information is known concerning them that, were the information we have to be applied, a huge amount of the nuisance complaints and disorders could be avoided. The knowledge we have concerning the relationship of

diet, exercise, rest and motivation, if applied from birth onward, could bring about a solid reduction in the amount of sickness and loss of time from work.

Science and medical practice are undergoing an important and healthy transgression. No longer should the doctor limit himself to curative procedures only. For there is the need for prevention of many diseases. And further, there is the possibility now of developing a new and finer and more rugged body and mind. Today we have the 40,000 mile tire. We are well on the way to the 100-year heart and mind and spirit.

With a dramatic lengthening of the life span, we must avoid all methods possible which create large numbers of medicated survivors. This emphasizes the importance of the basic needs of the elderly. They need to be kept within the stream of meaningful family and community living. No longer can the healthy older citizen be regarded as supernumerary. For too long the oldsters have been existing in a hostile society. They need to belong; they need to understand each has an important assignment in the social fabric. In this way they can continue to grow and enjoy a more meaningful, later period. Indeed, there are many healthy and well-adjusted older citizens who might make up a very excellent Peace Corps. Why should we send youngsters with but an introductory contact with life to foreign lands where there is need for understanding and give-and-take on the part of our representatives when a Corps of mature citizens would much better serve the purpose.

There are many areas for productive employment for mature men and women. In this group you should find many of the finest and most qualified teachers, other professional men and women, artisans, engineers, writers, woodworkers, florists, landscape gardeners and a whole host of areas where there is a need for personnel.

Personally, it is my opinion that Communism is not our great menace. Rather it is a shortening of and a deterioration of the physical and moral fiber of our American people. Today we are regarded as the richest and most powerful nation in the world. Will we still be so regarded in 1970? For a nation that has been

\* Abstracted from a paper given at the annual meeting of the Maine Conference of Social Welfare, Portland, Nov. 17, 1961

\*\* Senior Consultant of Medicine, The Lenkenau Hospital, Philadelphia

more than blessed with all of the material comforts of life do we not have a corresponding obligation and responsibility to other less favored nations? To make the most of our fabulous potentials we should have an energetic and effective educational and physical fitness program that will open up channels for development, growth and maturation for all the various segments of our population. The only way this can be done, as I see it, is to collect all of the material pertinent to the various major issues and then, from the information so gleaned, organize a long-range program which will open up more opportunities for continued growth and enjoyment of living throughout the entire life span. There is an upward thrust in the lives of all individuals; and now in the 60's and 70's many citizens, heretofore regarded as finished, are gaining their second wind. It is with knowledge, experience and understanding that they may be effective participants in the creation of a more rugged and finer way of life. This is the great promise of American citizenship.

At the present time there is a great amount of propaganda going on concerning medical care for older citizens. Certain elements of labor decry the sad state of our elders who have no money and are practically cast off by society. Many of my patients in the labor force are not so concerned, they tell me, about medical care as they are that labor has thrust them out and has forbidden them a job when they are perfectly capable of working.

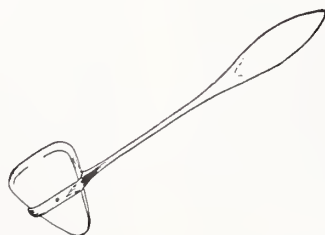
May I call to your attention three important publications. The first is "What Price Medical Care?" by Sir Earl Paige. This is published by Lippincott of Philadelphia. It describes in some detail the insurance program so successfully worked out by the citizens of Australia. Instead of waging war against each other the citizens with the government, voluntary and private insurance agencies, management, labor and the medical profession sat down around the table and worked out a program which might with good reason be an ideal answer to our medical care program in the United States.

As you know for many years Great Britain has had

a National Health Service. May I urge that you read two reports on this service. The first is "The Genesis of the National Health Service" by John and Sylvia Jewkes, published by the Blackwell Co. of Oxford, England. The other is the medical report of the Fellowship for Freedom in Medicine, Bulletin No. 48. If you will carefully study these three documents, you will have qualified answers to many of the questions now being raised by the good people of our nation. The time has come when, instead of one large and influential group trying to vilify other groups and discredit them, which only muddles the issues at stake, all groups should be willing to sit down around the conference table. The major questions can be answered. Before any bill is legislated into the law of the land, it is of the utmost importance that every citizen have all the facts. Then, with an intelligent and informed public, the solution will be forthcoming.

American medicine is dedicated to the highest ideals of citizenship. In fact, I would say that, as goes medicine, so will go American democracy. I can say with confidence that any program that will improve the health of individuals and families and communities will receive the militant support of the entire medical profession. Doctors, in addition to their professional responsibilities, also have a basic obligation as citizens. In these two areas we have many times been at fault. We are at all times willing to adjust our program and our policy when the indications are present. In the light of the experiences of other nations each citizen should be careful. When high pressure groups are in process of organizing a socialistic stampede, endeavoring thereby to force the law makers of our land to legislate an unwise proposal, an aroused and informed great American public should stand firm.

None of the proposals, and this is my candid opinion, are as yet satisfactory. But there is light on the horizon. With cooperation, with more light and less heat, we, as a great and sovereign people, are capable of finding a solution to the problems now facing us. This is the best guarantee for our American heritage.




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Secretary, Donald L. Anderson, M.D., Lewiston

## AROOSTOOK

President, Harry M. Helfrich, Jr., M.D., Presque Isle  
Secretary, Clyde I. Swett, M.D., Island Falls

## CUMBERLAND

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Secretary, Albert Aranson, M.D., Portland

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Secretary, Philip B. Chase, M.D., Farmington

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Secretary, Mustafa V. Onat, M.D., St. George

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Secretary, Albert P. Royal, Jr., M.D., Rumford

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Secretary, Frederick C. Emery, M.D., Bangor

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Secretary, Harland G. Turner, M.D., Norridgewock

## WALDO

President, Ward A. Albro, M.D., Belfast  
Secretary, Seth H. Read, M.D., Belfast

## WASHINGTON

President, Rowland B. French, M.D., Eastport  
Secretary, Karl V. Larson, M.D., East Machias

## YORK

President, Marcel D. Ouellette, M.D., Sanford  
Secretary, C. W. Kinghorn, M.D., Kittery

## County Society Notes

## KNOX

December 12, 1962

The Knox County Medical Association met at the Knights of Columbus Hall in Rockland, Maine on December 12, 1961.

The following officers were elected for the coming year:

President, William A. McLellan, M.D., Camden

Vice-President, John A. Root, M.D., Rockland

Secretary-Treasurer, Mustafa V. Onat, M.D., St. George

The guest speaker of the evening was Heinz Magendantz, M.D., of the New England Medical Center, Boston, whose topic was "Management of Cardiac Arrhythmias."

MUSTAFA V. ONAT, M.D.  
*Secretary*

January 9, 1962

The Knox County Medical Association met at the Knights of Columbus Hall in Rockland, Maine on January 9, 1962.

Earle M. Davis, M.D. of Waterville, guest speaker of the evening, spoke on "Common Urological Problems."

Henry O. White, M.D. of Rockland was elected to membership in the society.

The following members were elected to the Board of Censors: David V. Mann, M.D.; Wesley N. Wasgatt, M.D. and John A. Root, M.D. of Rockland.

Delegates to the Maine Medical Association House of Delegates were elected as follows: Albert Hunter, M.D., Camden and Merrill J. King, Jr., M.D., Rockland. Alternate: Johan Brouwer, M.D., Rockland.

A change of the monthly meetings from the second Tuesday to the first Tuesday of each month beginning in March was approved.

MUSTAFA V. ONAT, M.D.  
*Secretary*

## PENOBSCOT

December 18, 1961

A meeting of the Penobscot County Medical Society was held at the Pilot's Grill, Bangor, Maine on December 18, 1961 with the President, Richard C. Wadsworth, M.D., presiding. There were forty members and guests present.

Following a social hour and dinner, the business meeting was held. The report of the Nominating Committee presented by John E. Whitworth, M.D., Chairman, which consisted of the following officers for 1962 was approved.

President, Clement S. Dwyer, M.D., Bangor

President-Elect, Allison K. Hill, M.D., Bangor

Secretary, Frederick C. Emery, M.D., Bangor

Treasurer, Benjamin L. Shapero, M.D., Bangor

Councilor, Richard T. Munce, M.D., Bangor (3 yrs.)

Richard C. Wadsworth, M.D. thanked the various committee chairmen for their good work and cooperation during the year and then turned the meeting over to the new President, Clement S. Dwyer, M.D., who made appropriate acceptance remarks.

William P. Rogers, Jr., M.D. of the Pondville and Massachusetts General Hospitals was the speaker. Dr. Rogers spoke on the subject of Thyroid Cancer and showed several slides at the end of his talk. Asa C. Adams, M.D., Richard C. Wadsworth, M.D. and Lawrence M. Cutler, M.D. discussed various aspects of Thyroid Cancer following Dr. Rogers' talk.

PHILIP B. THOMAS, M.D.  
*Secretary*

## HANCOCK

January 10, 1962

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on January 10, 1962.

The meeting was opened by the President, James H. Crowe, M.D. Minutes of the previous meeting were read and approved.

Dr. Crowe was elected to appoint a Manpower Mobilization Committee. This committee will consist of Raymond E. Weymouth, M.D. and Silas A. Coffin, M.D. of Bar Harbor and W. Edward Thegen, M.D., Bucksport.

William H. Austin, M.D., Research Fellow at the Maine Medical Center in Portland, presented an interesting illustrated talk on blood dialysis and the uses of the artificial kidney. A lively question and answer period followed Dr. Austin's presentation.

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## YORK

January 10, 1962

Twenty-seven members and three guests were present at the York County Medical Society meeting which was held at the Henrietta D. Goodall Hospital in Sanford, Maine on January 10, 1962.

The meeting was called to order by the President, Kenneth E. Leigh, M.D., following a social hour and dinner.

James A. MacDougall, M.D., President of the Maine Medical Association, gave a very fine talk on state association matters. Thomas A. Martin, M.D., Councilor for the First District, discussed council activities in a very interesting manner.

Carl E. Richards, M.D., Chairman of the Nominating Committee, presented his report and the following officers were elected for the coming year:

President, Marcel D. Ouellette, M.D., Sanford  
Vice-President, James S. Johnston, M.D., York Harbor  
Secretary-Treasurer, Charles W. Kinghorn, M.D., Kittery  
Executive Committee, Drs. Ouellette, Johnston, Kinghorn,  
Paul S. Hill, Jr., and Melvin Bacon  
Councilor, Robert F. Ficker, M.D., Kennebunkport  
Board of Censors: Stephen A. Cobb, M.D., Sanford;  
Willard H. Bunker, M.D., York Harbor and Paul S.  
Hill, Jr., M.D., Saco

Delegates to the Maine Medical Association House of  
Delegates: Robert F. Ficker, M.D., Kennebunkport;  
Roger J. P. Robert, M.D., Saco and Carl E. Richards,  
M.D., Sanford. Alternates: Kenneth E. Leigh, M.D.,  
York; Stephen A. Cobb, M.D. and Melvin Bacon, M.D.,  
Sanford

The annual report of the Secretary-Treasurer was read and approved. Charles S. Turville, M.D. of Alfred was elected to membership in the society. Resolutions on the death of Herbert E. Locke, Esq. were read.

CHARLES W. KINGHORN, M.D.  
*Secretary*

## FRANKLIN

January 15, 1962

The Franklin County Medical Society met on January 15 and elected the following officers for 1962:

President, Gaetano T. Fiorica, M.D., Chisholm  
Vice-President, Stanley B. Covert, M.D., Kingfield

Secretary-Treasurer, Philip B. Chase, M.D., Farmington  
Delegate to the Maine Medical Association House of  
Delegates: Wallace H. Duffy, M.D., Farmington. Al-  
ternate: Paul E. Floyd, M.D., Farmington

Censor: Hays G. Bowne, M.D., Farmington (1 yr.)

James A. MacDougall, M.D., President of the Maine Medical Association, was the guest speaker of the evening. There was a very stimulating and enthusiastic discussion of current medical economic problems, particularly with relation to the proposed care for the aged plans.

PHILIP B. CHASE, M.D.  
*Secretary*

## LINCOLN-SAGADAHOC

January 16, 1962

A meeting of the Lincoln-Sagadahoc County Medical Society was held at the Ledges, Wiscasset, Maine on January 16, 1962. Sixteen members and one guest were present.

Sidney C. Dalrymple, M.D. of South Lincoln, Massachusetts was elected to membership in the society.

The following officers were elected for the year 1962:

President, Hamdi Akar, M.D., Bath  
Vice-President, Ralph C. Powell, M.D., Damariscotta  
Secretary-Treasurer, George W. Bostwick, M.D., Newcastle  
Delegates to the Maine Medical Association House of  
Delegates: Ralph C. Powell, M.D., Damariscotta and  
John F. Andrews, M.D., Boothbay Harbor. Alternates:  
Mary J. Tracy, M.D., Damariscotta and Miriam Doble,  
M.D., Bath

Board of Censors: Samuel L. Belknap, M.D., Damariscotta;  
John F. Dougherty, M.D. and Virginia C. Hamilton,  
M.D., Bath

Philip L. Archambault, M.D., Orthopedic Surgeon of Lewiston, was the guest speaker of the evening.

GEORGE W. BOSTWICK, M.D.  
*Secretary*

## CUMBERLAND

January 18, 1962

A meeting of the Cumberland County Medical Society was held at Valle's Steak House in Portland, Maine on January 18, 1962. One-hundred and seventy seven members and guests were present. Guests present included: Mr. John F. Kiser, Field Representative of the American Medical Association; members of the Woman's Auxiliary to the Cumberland County Medical Society; as well as interns and residents of the Mercy Hospital and Maine Medical Center and their wives.

Following a social hour and dinner, a film was shown entitled, "On Call To The Nation." This film had been produced by the British Broadcasting Company in order to demonstrate the results of the first ten years of socialized medicine in England.

ALBERT ARANSON, M.D.  
*Secretary*

## KENNEBEC

January 18, 1962

Thirty-six members were present at the meeting of the Kennebec County Medical Association which was held at the Worster House in Hallowell on January 18, 1962.

John D. Denison, M.D. of Gardiner was elected as a mem-



Loring W. Pratt, M.D., President Kennebec County Medical Association and Clement A. Hiebert, M.D., guest speaker.

ber of the Council to replace Hugh J. Mathews, Jr., M.D. of Gardiner who resigned.

Clement A. Hiebert, M.D. of Portland presented a paper entitled, "Precordial Pain and the Incompetent Gastric Cardia."

EARLE M. DAVIS, M.D.  
*Secretary*

### New Members

#### ANDROSCOGGIN

Gerard L. Morin, M.D., 104 Ash Street, Lewiston

#### CUMBERLAND

A. Dewey Richards, M.D., 11 Gage Street, Bridgton

#### KENNEBEC

Joseph P. Senenkyj, M.D., P. O. Box 724, State Hospital, Augusta

#### KNOX

Henry O. White, M.D., 22 White Street, Rockland

#### LINCOLN-SAGADAHO

Sidney C. Dalrymple, M.D., So. Great Road, So. Lincoln, Massachusetts

#### WASHINGTON

Israel Schlain, M.D., Jonesport

#### YORK

Charles S. Turville, M.D., P. O. Box 187, Alfred

### Deceased

#### CUMBERLAND

C. Earle Richardson, M.D., 3 Cumberland Street, Brunswick Maine, December 17, 1961

#### AROOSTOOK

Loren F. Carter, M.D., 33 1st Rangeway, Waterville, Maine, January 25, 1962

#### PENOBSCOT

Henry C. Knowlton, M.D., 245 Center Street, Bangor, Maine, January 7, 1962

## News, Notes and Announcements

**State of Maine Board of Registration of Medicine  
Secretary — Daniel F. Hanley, M.D.,  
Brunswick, Maine**

**Physicians Licensed to Practice Medicine and  
Surgery in the State of Maine  
November 14-16, 1961**

#### THROUGH EXAMINATION

Rodrigue J. Albert, M.D., St. Francis Hospital, Hartford, Connecticut

Bahram Bahrami, M.D., 2601 Carondelet Street, New Orleans, Louisiana

Hemendra N. Bhatnagar, M.D., 22 Morrill Avenue, Waterville, Maine

Adrian V. Blake, M.D., Box C, Waverley, Massachusetts

Thomas F. Conneen, M.D., Lafayette Hotel, Portland, Maine

Allan B. Easton, M.D., 231 East Prospect Avenue, Mount Vernon, New York

Ihsan Egeli, M.D., 86 East 49th Street, Brooklyn, New York

Sherif Shafey Mohamed El-Shafey, M.D., 665 New York Avenue, Brooklyn, New York

Firooz Emami, M.D., Lahey Clinic, Boston, Massachusetts

Behzad Fakhery, M.D., Memorial Center, New York, New York

Donald C. Finlayson, M.D., 104 Indian Head Road, Framingham, Massachusetts

Leandre W. Giguere, M.D., Springfield Hospital, Springfield, Massachusetts

George R. Hug, M.D., The Children's Hospital, Cincinnati, Ohio

David M. Iszard, M.D., Lemuel Shattuck Hospital, Jamaica Plain, Massachusetts

Mohammad Rafiq Jan, M.D., Medfield State Hospital, Hard- ing, Massachusetts

Rostam S. Khorsandian, M.D., Philadelphia General Hospital, Philadelphia, Pennsylvania

Hakkew Kim, M.D., New England Deaconess Hospital, Boston, Massachusetts

Bernhard Laukenmann, M.D., 3106 Hamilton Avenue, Baltimore, Maryland

Hans Leen, M.D., Medical Center Staff House, Jersey City, New Jersey

Hsing C. Loh, M.D., Yale University School of Medicine, New Haven, Connecticut

Luis A. Marco, M.D., 1601 West Taylor Street, Chicago, Illinois

Jack H. Marcovitch, M.D., 48 McCarthy Road, Newton Centre, Massachusetts

Leonardo R. Martinez, M.D., 153 East 49th Street, Brooklyn, New York

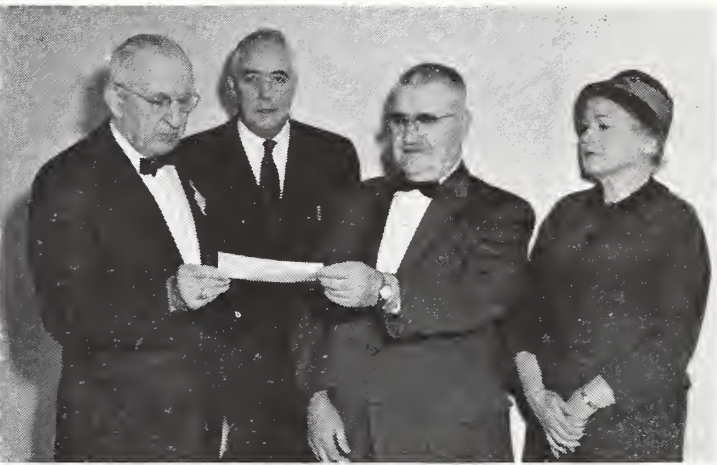
Pompeyo B. Montemayor, M.D., 444 East 68th Street, New York, New York  
H. Padmanabhan, M.D., Yale University School of Medicine, New Haven, Connecticut  
Giuseppe G. Pietra, M.D., Massachusetts General Hospital, Boston, Massachusetts  
Jose M. Rodriguez, M.D., 5806 Fifth Avenue, Pittsburgh, Pennsylvania  
Remedios K. Rosales, M.D., 5023 Hazel Avenue, Philadelphia, Pennsylvania  
Ferdinand A. Rossmann, M.D., St. Barnabas Hospital, New York, New York  
Yung H. (Joseph) Son, M.D., The Ontario Cancer Institute, Toronto, Canada  
Alexander G. Sterkevych, M.D., Lahey Clinic, Boston, Massachusetts  
Guy St.Pierre, M.D., Le Centre Medical, Edmundston, N.B., Canada  
Alberto E. Trentalance, M.D., The Cooper Hospital, Camden, New Jersey  
Richard W. Turcotte, M.D., The Springfield Hospital, Springfield, Massachusetts  
Wang-Yen, M.D., Jefferson Medical College Hospital, Philadelphia, Pennsylvania

THROUGH RECIPROCITY

Noel Henry Aldridge, M.D., Massachusetts General Hospital, Boston, Massachusetts  
Jack Bocher, M.D., U. S. Navy Security Group Activity, Winter Harbor, Maine  
Robert B. Bunker, M.D., 655 High Street, Westwood, Massachusetts  
Raymond L. Candage, M.D., 55 Appletree Lane, Holden, Massachusetts  
Frederick W. Cheney, Jr., M.D., 109 Montrose Avenue, Portland, Maine  
Rahim Farid, M.D., P. O. 108, Brazil, Indiana  
Frank Fodor, M.D., P. O. Box 271, Petersburg, Virginia  
M. Taghi Ghavamian, M.D., 720 South Wolcott, Chicago, Illinois  
Richard N. Goldman, M.D., 208 South Huntington Avenue, Jamaica Plain, Massachusetts  
Lyman H. Hoyt, M.D., 51 Bay State Road, Boston, Massachusetts  
Sung J. Liao, M.D., Curtis Road, Middlebury, Connecticut  
Benigno A. Manubay, M.D., Veterans Administration Hospital, Rutland Heights, Massachusetts  
Alejandro D. Paniagua, M.D., New Jersey State Hospital, Ancora, New Jersey  
Lawrence Rothstein, M.D., 725 South Eastmoor Boulevard, Columbus, Ohio

Edward T. Schnoor, M.D., P. O. Box 313, York Harbor, Maine  
Franz Schubert, M.D., Route 5, Rio Road, Charlottesville, Virginia  
Gilbert L. Shapiro, M.D., 75 Francis Street, Brookline, Massachusetts  
Abdol Hamid Shojania, M.D., Sunny Acres, Richmond Road, Cleveland, Ohio  
Umberto Squattrito, M.D., Connecticut Valley Hospital, Middletown, Connecticut

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## Book Reviews

**Differential Between Normal and Abnormal in Electrocardiography** — By Ernst Simonson, M.D., 7<sup>o</sup>, Cloth, pp. 328, with numerous tables, charts, and cardiograms. St. Louis: The C. V. Mosby Company, 1961. \$13.50.

Despite recent advances or changes in electrocardiographic theory, the interpretations of tracings remains quite empirical. The interpreter is faced daily with the dilemma of minor or borderline changes to label "within normal variation" or "abnormal." No doubt, the problem will always exist, but one can find considerable assistance in this volume. The author and his colleagues, using a representative adult population sample, have compiled tables which define reasonable limits (include 95% of "normals") for various amplitudes and intervals, and relates them to age, body weight, sex, chest configuration, etc. There have been scattered reports in the literature dealing with various aspects of this problem. But here, for the first time in a single volume, appear complete tables together with helpful discussion. As mentioned, these tables deal with adults. (Ziegler published a similar work dealing with the pediatrics age group in 1951.) The necessary statistical discussions, always confusing to the uninitiated, are presented clearly. There are informative sections on the various stress tests and spatial vectorcardiography.

Although the book is not a textbook or primer, it has value other than as a reference or atlas. There are helpful points which do not lend themselves to tables or measurements, dealing with qualitative changes. There is an excellent bibliography. I believe this book fills a definite need. It will be of limited use to the beginner or occasional cardiographer, but of distinct use wherever tracings are read with any frequency. The information it contains is nowhere duplicated. The problem of "abnormal" vs "normal" in an individual tracing will

persist, but the interpreter can be bolstered by a statistical aid which will at least indicate the probability.

ROBERT G. MOHLAR, M.D.  
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**Progesterone and the Defense Mechanism of Pregnancy** — Published by Little, Brown & Company, Boston, February, 1961, pp. 108.

This book is a transcript of a Ciba Foundation Study Group Symposium which took place in London in 1961. The main article in the book is presented by Dr. Arpad Csapo of the Rockefeller Institute in which he fairly thoroughly develops his theory of the so-called progesterone block on the pregnant myometrium. The basic facts presented concern mainly phenomena observed in lower animals in which the placental progesterone appears to act locally on the myometrium over the placenta rather than systemically on the entire uterine muscle. Labor occurs only when the activity of the rest of the myometrium overcomes the local progesterone inhibiting effect at the placental site. Dr. Josef Zander of Cologne presents evidence that the fetus itself receives about half of the progesterone produced by the placenta and metabolizes it. This is shown by differential assays of umbilical venous and arterial blood. The umbilical artery, for instance, carries only about 1/3 of the progesterone present in the umbilical vein. The umbilical artery, however, does have more of other progesterone metabolites than the vein.

Other chapters concern the myometrial cell, electrolytes, and electrophysiology of the uterus. After each paper there are good discussions by the panel in which many recently discovered facts are divulged.

STANLEY W. KENT, M.D.  
Portland, Maine

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## A Disease Of Many Names

(Benign Paroxysmal Peritonitis, Armenian's Disease, Familial Recurring Polyserositis, Periodic Disease, Familial Mediterranean Fever, Periodic Peritonitis, Periodic Abdominalgia)

CHARLES A. HANNIGAN, M.D. and RUDOLPH HAAS, M.D.\*

This is a case report of an obscure disease which will remain obscure until the medical profession, particularly the surgical segment, becomes completely aware of its existence. Part of the obscurity is due to its many names. Physicians describing it have named it for features that appeared most outstanding to them, more or less as the fabled blind men described the elephant.

There are two important reasons for knowing about Benign Paroxysmal Peritonitis. It can mimic an acute surgical abdomen and should be included in the differential diagnosis of the acute abdomen. Also, there are now indications that it is treatable by a low fat diet, thus eliminating the mental and physical pain the patient must endure, as well as possibly preventing the serious sequella of renal amyloidosis.

The excellent original report of Dr. Siegal in 1945 describes this disease and its clinical picture. Following is the introduction of Dr. Siegal's paper on Benign Paroxysmal Peritonitis.

"The purpose of this paper is to describe in detail an unusual clinical syndrome which is at present little understood and often undiagnosed. The characteristics of this disorder are constant and distinctive.

"The syndrome is characterized by recurrent paroxysms of severe abdominal pain with fever which may be as high as 105° F. Chilliness or a shaking chill may accompany the attacks. Involvement of the peritoneum is indicated by the subjective symptom of marked ab-

dominal soreness and the objective finding of widespread, exquisite direct and rebound tenderness. On occasion true involuntary spasm of the abdominal wall may be noted. These abdominal signs are often so striking that to the surgeon they suggest an acute abdominal peritonitis lesion. Emergency operation has been repeatedly urged.

Chest pain of a pleuritic type is frequently present at some stage of the attack. Marked malaise, severe prostration and intense nausea and vomiting are almost constant characteristics. Diarrhea is conspicuously absent. Leukocytosis is a frequent finding. This disease affects young people, often beginning in the second or third decade and continuing for many years. Nevertheless they remain in good general health and their disorder, essentially benign, continues without the development of any persistent anatomical lesion and without permanent impairment of any physiologic function."

He also mentioned that there was occasionally joint involvement in this syndrome. Since his paper, there have been several other papers emphasizing certain aspects of the disease and naming it after them. Confusion has resulted. As a prime example, the table of contents of the July, 1961 issue of the Archives of Internal Medicine lists the disease differently three times: Familial Mediterranean Fever, Recurrent Polyserositis, and Periodic Disease.

Dr. Reimann in 1948 included this particular disease as a Periodic Disease, along with Periodic Fever, Cyclic Neutropenia and Intermittent Arthralgia.

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According to Heller, among 253 cases available in the world literature up to 1958, 85% of the patients whose origin was known were either Jews or Armenians; 159 were Jews; 58, Armenians; 24, Lebanese Arabs; 1, Syrian Arab; 2, Israeli Arabs; 2, Iraqi Arabs; 1, Turk; 1, Italian. For five, no data was available. Of the Jews, all of Heller's patients were Non-Ashkenazi Jews. The disease is most prevalent among those people living in the Mediterranean area. It is obvious why it is called Armenian's Disease; and, why it is called Familial Mediterranean Fever.

Others have called it Familial Recurrent Polyserositis or Recurring Polyserositis. Priest and Nixon reported 20 cases under the title of "Familial Recurring Polyserositis: A Disease Entity." They added another name to the literature to broaden the clinical symptomatology to include any of the definite serous cavities, but still with the characteristic fever, pain and elevation of white count.

Our review of the literature certainly indicates that there is a definite disease entity and that a good deal of the confusion and lack of recognition of the syndrome probably results from the variety of terminology. We do not wish to add further to the confusion, but do suggest returning to Dr. Siegal's original name. Benign Paroxysmal Peritonitis certainly is a descriptive phrase and should alert the surgeons to include it in the differential diagnosis of the acute abdomen. The disputed word "benign" is in contrast to the urgent implications of the usual peritonitis.

It is reasonable to expect that normal appendices will be removed in patients developing this condition for the first time and having mainly right lower quadrant pain with direct and rebound tenderness. However, the recurrence of symptomatology following appendectomy in a patient of Mediterranean background, particularly Armenian or Non-Ashkenazi Jew, with possibly a family history of others with a similar difficulty, should lead to a correct diagnosis.

The following is a case presentation which we feel is typical of Benign Paroxysmal Peritonitis in its clinical course and the long interval between onset of the disease and final recognition.

This 38-year old white male of Armenian origin stated as his chief complaint attacks of abdominal pain and fever.

He was born in Revere, Massachusetts. In his childhood, he had had frequent attacks of severe infrascapular pleurisy. He had been in the Army for several years in India and Australia and had been on antimalarials. Aside from several episodes of diarrhea, his tour of duty was characterized by good health.

In 1946, he first entered the Central Maine General Hospital with a chief complaint of pain in the abdomen for 40 hours, with nausea and vomiting. He had begun to have generalized, crampy pain in the abdomen 40 hours prior to admission, which gradually increased over a 6-hour period, until he had to stop work. Later

on, there was nausea and vomiting. The pain was worse the day of admission, but there was no localization of the pain. On admission, his white count was 13,200 with a differential of 58% polys., 27 lymphs., 12 stabs, 3 monos. Examination of the abdomen after morphine showed a flat abdomen with no masses or tenderness. Laparotomy for acute appendicitis revealed a retrocecal appendix, which was removed.

The pathologist reported marked hyperplasia of the lymphoid tissue, slight eosinophilia, slight fibrosis of the inner muscle layer. The diagnosis was sub-acute appendicitis.

For the next seven to eight years, he had abdominal pain off and on, usually subsiding within 12 hours. In May, 1957, he was next admitted to the Central Maine General Hospital because of severe abdominal pain of 36 hours' duration, which was diffuse at first and then localized in the left lower quadrant.

Physical examination showed marked tenderness in the entire LLQ with a question of a palpable mass. No peristalsis was noted in that area. His attending physicians considered volvulus and ureteral colic in the differential diagnosis. There were 5 to 25 white cells per high power field in the urine sediment. IVP was negative. The pain and tenderness in the LLQ persisted. Temperature was normal; white count, 11,800; differential, 74 mature polys., 19 lymphs., 4 monos., 3 stabs.

At laparotomy, dense adhesive bands between the adjacent loops of the terminal ileum and additional adhesive bands in the cul-de-sac between the peritoneal surfaces and the sigmoid were found. Following this, the patient had other episodes of abdominal pain and was readmitted in September, 1957, because of abdominal pain of unexplained origin. Two weeks prior to this admission, he again developed an attack of severe abdominal cramps, which required large amounts of opiates. This attack lasted two days. Urinalysis was normal; white count, 8,800; 49 mature polys.; 43 lymphs.; 3 monos.; 4 stabs; 1 eosinophil; sedimentation rate, 21 mm./h.; VDRL test for syphilis was negative; barium meal, normal; chest x-ray, normal; Graham Series, normal; barium enema, normal. The patient was discharged to be followed in the office.

Because of continuing difficulty, he was referred in 1958 to a clinic in Boston for evaluation. Gastric analysis was within normal limits. Urine tests for porphobilinogens, uroporphorins and coproporphyrins were negative. An oral cholecystogram revealed a normally functioning gallbladder. Hemoglobin, 15.7 gm.%; hematocrit, 48%; white count, 8,650; serology, negative; sedimentation rate, 20 mm. per hour; FBS, 79 mg.%; NPN, 31 mg.%; bilirubin, 0.1 mg.%; transaminase, 15 units; alkaline phosphatase, 3.3 Bodansky units. An x-ray of the chest was normal, as was barium enema, barium meal and serial films of the small bowel. Lateral abdominal films did not reveal any evidence suggestive of internal hernia.

It was of interest to his physicians that the gall-

bladder pills seemed to bring on an attack of distress, accompanied by diarrhea. In retrospect, this may have been due to the fatty meal.

He was discharged on antispasmodics and a bland diet. However, 3 months later because of recurrent distress, he was readmitted. A spinal fluid examination was normal. EKG, IVP, upper GI series and small bowel studies were again normal. Despite a normal electroencephalogram, a trial of Dilantin, 100 mg. t.i.d. was instituted. However, he continued to have symptoms and on August 15, 1958 an exploratory laparotomy was done; this was negative except for peritoneal adhesions.

The patient continued to have attacks of abdominal pain with fever as high as 103 degrees. The patient stated that usually the pain would occur as an aching sensation just below the umbilicus, would increase in intensity and finally localize in an area just to the left of the umbilicus, where there was direct and rebound tenderness. At times, there was vomiting. Later on, the pain would seem to move into the RUQ with aggravation by respiration. The 24-hour excretion of lead was 0.007 mg.; porphobilinogens were negative. During these attacks, urinary diastases had ranged from 250 to 329 units. The patient required opiates to relieve his pain. On one occasion, a diagnosis by x-ray of paralytic ileus was made. The patient became increasingly distressed, as the attacks occurred as often as once a month.

Finally, in the summer of 1961, a diagnosis of Benign Paroxysmal Peritonitis was made on the basis of the characteristic clinical picture. He was put on a 20 gram fat diet, and also told to take 100 mg. of Prednisone at the first sign of an attack. After two months on this diet, he developed the prodrome of fever and abdominal soreness. He took 100 mg. of Prednisone and felt that for the first time the pain of the attack had been aborted. He was then tapered off Prednisone and had recurrence of pain, which required Demerol®. He refused further Prednisone and after two days recovered spontaneously. He felt the Prednisone had prolonged the attack, although it had definitely aborted the pain. It was then learned that, through a misunderstanding, he had not been following the strict 20 gram diet, as had been supposed.

Since going on the 20 gram fat diet, he has been without symptoms. Admittedly, the duration on this strict diet has been only three months. Also, the patient says the diet is very difficult to follow and admits he occasionally exceeds his daily fat quota.

The family history, as obtained from his sister at a later date, was quite interesting and is reported almost verbatim.

It seems that in the family it was generally known

and particularly by the mother that the children would often have attacks of chest or abdominal pain. She knew that if they got pain in the shoulder, they would have trouble breathing. But, it might hit them in the abdomen and there would be nausea and possibly fever. They would go to bed, take aspirin, and in a day or so be better. The girls felt that this was particularly bad if they became excited or upset. The mother told them that they would get over it at 40. The oldest sister and the youngest sister had had their appendices removed. The sister, aged 45, who gave me this information had not.

## DISCUSSION

If one is aware of the syndrome of Benign Paroxysmal Peritonitis and encounters a case, there should be no problem in establishing the diagnosis on clinical grounds alone. It occurs in people of Mediterranean origin. It is familial. There is fever, either a definite peritonitis or pleurisy. There is usually leukocytosis. At laparotomy, exudate has been found with localized peritonitis. If the disease continues unabated, there is good evidence that it is not benign and that amyloidosis with renal failure will develop.

At present, the treatment which merits most consideration is the 20 gram low fat diet, even though it may be very difficult to follow. Apparently, steroids abort the acute attack.

In summary, a patient with Benign Paroxysmal Peritonitis has been presented. Diagnosis was made on the basis of the distinctive clinical picture. We feel that the multiplicity of terms is adding greatly to the confusion associated with this particular disease and suggest that the name "Benign Paroxysmal Peritonitis" be retained.

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# Islet Cell Tumor Of The Pancreas

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During the past 40 years great progress has taken place in our knowledge of the pancreas. In 1922 Banting and Best confirmed the endocrine activity of islet cells and demonstrated that the internal secretion — insulin — would control diabetes. The first functional carcinoma, with insulin-containing liver metastases, was reported in 1927 by Wilder et al. The first surgical cure of hyperinsulinism was reported by Dr. Roscoe Graham who removed a localized islet tumor in 1929. By 1930, two other cases had been reported — both with autopsy findings. A review of the literature in 1935 by Drs. Frantz and Whipple revealed only 25 cases of adenoma of islet cells with hyperinsulinism. By 1940 there were ninety-six reported cases, and by 1949 there were 258 functional tumors reported in the literature. In the following eight years, however, there were 81 additional cases reported by Porter and Frantz; a total of 339 cases up to 1957.

Islet cell adenomas occur about once in one thousand autopsies. Clinical manifestations are apparent in about 20% of these. They are equally distributed among males and females and they have been observed at almost every age, ranging from 6.5 weeks to 84 years. The average age appears to be about 40 years. Ninety percent of the individuals under thirty years of age found to have islet cell adenomas also have severe hypoglycemia. By the same token, hyperfunctioning islet cell adenomas are rarely seen in patients past sixty years of age. The size of islet cell tumors may vary from 1 mm. to 15 cms. with most of them measuring 1 cm. to 2 cms. There is no relationship between the size of the adenoma and the degree of symptoms. Islet cell adenomas occur more commonly in the tail of the pancreas.

Grossly, islet cell adenomas are round, firm, discrete tumors which are circumscribed and usually encapsulated. The surface is more vascular than the adjacent tissue. If the tumor is in the substance of the gland it is firmer than the remainder of the organ and its cut surface is more homogeneous in appearance. The presence of degenerative changes in the adenoma may alter its color and consistency, particularly if calcification is present. Calcification may occur without diminishing the physiological activity of the functioning adenoma.

Microscopically, these tumors appear as massive islets of Langerhans with relatively normal internal

architecture. Individual variations in cellular patterns are observed in individual tumors as well as in the same tumor. No significant gross or microscopic variations are sufficient to explain the presence or absence of hyperinsulinism.

The symptoms of islet cell neoplasms depend on whether the tumors are insulin-producing or non-functional. The benign islet cell tumors which are not insulin-producing are largely chance findings as tiny tumors at autopsy.

The symptoms of insulin-producing tumors are those of spontaneous hypoglycemia. In order to rule out other causes of a faulty carbohydrate metabolism, the clinician must be alert to the possibility of hepatic disease, anterior pituitary hypofunction, and adrenocortical hypofunction. After diseases of the liver, pituitary and adrenals have been excluded, it becomes quite apparent that the nervous manifestations which are characteristic of the state of hypoglycemia are undoubtedly due to a pancreatic adenoma. The attacks tend to occur, however, at the same time of day — often before breakfast; although attacks later in the day may be related to greater activity which augments the effects of hunger. The Whipple Triad is well known and is as follows: (1) attacks which occur in the fasting state; (2) minimum blood sugar (less than 50 mgs%); and, (3) attacks immediately relieved by the administration of sugar. A provocative test of carbohydrate deprivation that results in a blood sugar value below 40 mgs./100 cc. is indicative of hyperinsulinism in most cases.

The treatment of islet cell tumors is, therefore, surgical. Islet cell tumors, as has been mentioned, are most frequently found in the tail and body of the pancreas and should be looked for there first. If a tumor is not seen on the anterior surface, the peritoneal attachment along the inferior border of the pancreas should be divided and by blunt dissection and elevation the posterior surface of the body and tail should be inspected. If none is seen, the gland should be carefully palpated. The islet cell tumors feel like discrete nodules — firmer than the surrounding pancreatic tissue — even when imbedded in the body and head of the organ, and are unmistakable. If no tumor is seen or felt in the body or tail, the duodenum should be mobilized to the midline by incising the peritoneum along the curve of the duodenum. This gives access to the posterior surface and permits the essential and free palpation of the head of the pancreas. If an adenoma is found, the same careful search must be made because more than one tumor may be

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FIGURE 1.  
Resected distal third of pancreas, (measuring 9 cms. long, 2.5 cms. diam., and weighing 22 gms.) Terminal portion split, dividing adenoma into two equal halves, each 1.5 cms. diam.

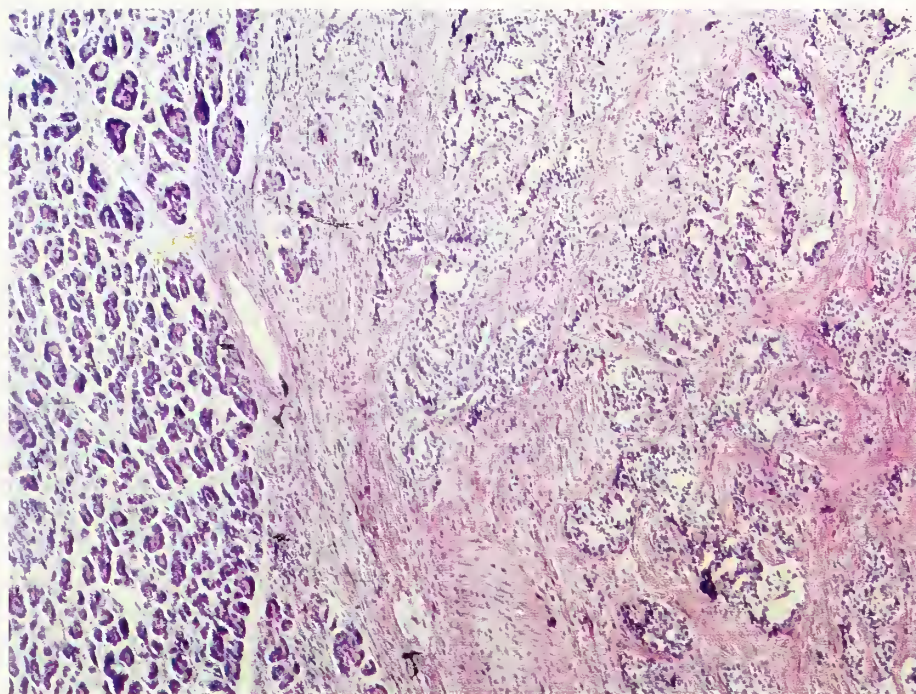
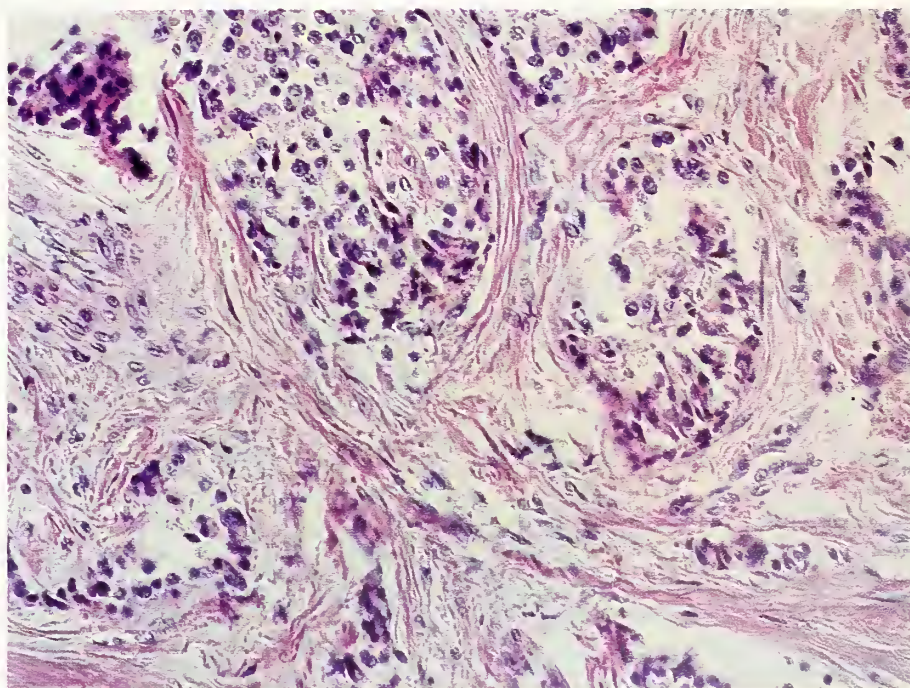


FIGURE 2.  
Junction of adenoma and normal pancreas. No true capsule but no invasive foci demonstrable. Tumor composed of double layers of islet cells, arranged in cords or about the periphery of potential spaces or islands. Exceptionally heavy, highly vascular stroma. H&E X100

FIGURE 3.  
High power detail of islet cells, showing their pattern of proliferation and strong tendency to form concentric masses akin to true islands of Langerhans. H&E X400





present. If no tumor is found after a very complete exploration, the decision as to resection will have to be made. In the cases where no tumor has been found in the body or tail (that is, that part of the pancreas to the left of the superior mesenteric vessels), a subtotal pancreatectomy may be done. The results have been better than with the removal of a small portion of the tail. The bed of the pancreas should be drained, after ligating the pancreatic duct. The use of fine silk for the smaller vessels and a heavier grade for the larger vessels will prevent the digestion of ligatures which occurs when catgut is used in pancreatic tissue. Howard, Morse and Rose found that simple enucleation of the adenoma relieved the symptoms in 86.4% of their cases. The mortality of their operation was 9.3%. The same authors found that of 77 cases who had subtotal pancreatectomies, because of failure of the surgeons to find a tumor, only 42.9% of this group obtained relief from this maneuver; but, an additional 9.1% were improved. Their mortality rate from subtotal resection was 14.3%. Of 46 patients in whom the removed portion of the pancreas was microscopically normal, 11 were later found to have an adenoma.

Postoperatively, almost all patients in whom a functioning adenoma has been removed have transient hyperglycemia for a period ranging from 1-14 days following the operation, but permanent diabetes rarely occurs — even after extensive pancreatic resection. Payne and Thorne found but two cases in which insulin was required for more than one month after operation. A temporary pancreatic fistula may occur but it usually closes rapidly and spontaneously. During the period of external drainage of pancreatic fluid, the wound is best cared for by constant suction.

#### CASE REPORT

This 78 year old female entered the CMG Hospital via ambulance on February 9, 1961, because of unconsciousness. She had never been seriously ill. Her usual weight was 134 lbs. She had routine physical examinations in 1954, 1955, and 1958 and appeared generally in good health. Her only complaint during these years was arthritis of the left knee.

For the first time in March of 1960, the patient experienced an attack of dizziness and weakness. She felt unsteady on arising and had little energy. She was examined at that time; her weight was 132 lbs., blood pressure 210/70, pulse 112. Physical findings and urinalysis were negative. The patient was given a mild sedative and was not seen for another year. By February, 1961, she complained of marked nervousness and weakness in her legs; she was excessively hungry, and on one occasion she fell "because her legs gave out." She had episodes where she felt "panicky." On examination her weight again was 132 lbs., blood pressure 170/70, pulse 90. Physical findings again were essentially negative.

Two days later the patient was found unconscious on

the floor of her bedroom at 5:00 P.M. and it was estimated that she might have been in this condition for 1 to 2 hours. When she was first found she seemed to start responding and was able to say a few words but within the next half hour she lost the ability to talk and had difficulty moving her left arm. She was taken to the hospital by ambulance.

Physical examination on admission revealed an elderly female patient who was adequately nourished. She was able to respond to questions, although somewhat slowly. Color was pale, skin was cool and dry. No definite facial paresis was noted. Tongue was moist and protruded in the midline. Blood pressure was 140/80, pulse 98, respirations 24. Heart, lungs and abdomen were essentially negative. There was marked peripheral arteriosclerosis. There appeared to be definite weakness of the left arm but reflexes were active and equal and Babinski was negative.

The patient's condition improved rapidly throughout the next 12 hours without treatment. She was completely alert the next morning and her speech was back to normal; however, she did not recall any events between 4 to 11 P. M. the previous day. The weakness of her left arm had completely subsided and reflexes were again entirely normal. Inasmuch as the difficulty in speech and the paresis of the left arm in a patient her age seemed to indicate a CVA, a lumbar puncture was done; normal spinal fluid pressure was found; 250 fresh red blood cells per cu. mm. were noted and the total protein was 68 mgs%. Blood count the morning after admission was essentially normal. Urinalysis was negative except for 1+ acetone. The NPN was 36 mgs% and the fasting blood sugar was 20 mgs%. Because of the latter findings a 2 hour post-prandial blood sugar was taken the same day and was found to be 34 mgs%. The next morning her fasting blood sugar was 39 mgs% and the CO<sub>2</sub> 27 meq/l.

A barium meal was done four days later but failed to reveal any abnormality in the area of the duodenal loop. On the basis of the above findings a laparotomy was decided upon and was carried out on February 16, 1961. The operative report follows: Examination of the gallbladder, stomach, spleen, kidneys and bowels was negative. After opening the gastrocolic omentum the entire pancreas was visualized. There was no tumor visible or palpable; however, the inferior margin of the pancreas was dissected free to the left of the midcolic artery and the body and tail of the pancreas were explored between thumb and index finger and only after this procedure had been done was a marble-sized solitary tumor palpable in the distal third of the pancreas on its superior-posterior margin near the tail. There were no other tumors palpable in the pancreas. The pancreas was transected at the junction of its middle and distal thirds and the transected pancreas was sutured with interrupted black silk. Gelfoam® was placed in the pancreatic bed. A latex drain was placed down to this area and brought out through a

stab wound to the left of the incision. The gastrocolic omentum was approximated and the wound was closed in anatomical layers with retention sutures in place."

The patient withstood the procedure very well and two hours after the completion of the operation her blood sugar was 207 mgs%. Six hours later blood sugar was 181 mgs% and the following morning it was 140 mgs%. It remained at that level during the next few days. Prior to discharge a 2 hour post-prandial blood sugar was 130 mgs%. Her urine remained free of sugar and acetone and the patient was discharged ambulatory on the 10th postoperative day.

Since that time the patient has been asymptomatic without insulin; however, she has not been able to maintain her pre-operative weight; she now weighs 110 lbs.

#### CONCLUSION

A brief summary of the literature and a case report of islet cell adenoma is presented. Even though this is a rare tumor, its identification and treatment is a rewarding one.

ACKNOWLEDGMENT: Appreciative acknowledgment is made to Dr. Charles F. Branch for the color photographs and accompanying legends.

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## Thrombocytopenic Purpura Due To Quinidine Case Report\*

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Drug reactions involving the blood forming organs are of great importance because of their potential seriousness. Agranulocytosis is perhaps the most frequent and severe type of reaction and can result from the sulfonamides, aminopyrine, thiouracils, butazolidine, gold and certain antihistaminics and antibiotics. Hemolytic anemia may result from the use of several types of drugs but has occurred more commonly after sulfonamide administration or exposure to naphthaline or acetanilid. Aplastic anemia is a less frequent but most serious toxic effect occasionally seen after use of chloramphenicol, gold, trimethadione, mesantoin and the sulfonamides.

Thrombocytopenic purpura may occur as a hemotoxic effect of sedormid, quinine, quinidine, thiouracils, mesantoin and the sulfonamides. Its appearance in each instance is often dramatic, heralded by a marked bleed-

ing tendency and amenable to treatment only when it is recognized as a drug-induced thrombocytopenia.

The purpose of this paper is to report an instance of quinidine purpura so that physicians will be alerted to this occasional toxic effect of quinidine.

#### CASE REPORT

This 63 year old housewife was admitted to the Central Maine General Hospital, 3/7/61, complaining of the sudden appearance of large black and blue areas over the left hip and both lower legs. For about a year she had been having recurrent attacks of rapid heart action, each of which would come on suddenly, last a variable period of time from a few minutes to several hours and finally stop abruptly. With these there was associated fullness and pressure in the chest with some pain radiation into the arms. During recent months she had been taking one 0.2 Gm. tablet of quinidine sulfate for these episodes of rapid pounding heart following

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which she would lie down and the attack would quickly stop.

On the day prior to this admission she had taken another one of the quinidine sulfate tablets for a recurrent attack of tachycardia which lasted for about 2 hours. Some 3 hours later, when she was up and about, there was onset of rather severe pain beginning posteriorly in the region of the left sacroiliac joint and extending around the hip and down the thigh laterally. This persisted, was quite severe, and later required a hypodermic injection of demerol for relief. During the night she had prolonged epistaxis for the first time. On the morning of admission she had noticed several large black and blue areas about the left hip and thigh and lower leg. She had also noted purple spots on her lips that were not present earlier. There had been no chill, fever or weight loss.

In the past, she had thought that she bruised more easily than the average person but she had never had a definite bleeding tendency. She had thought that this might be a familial trait since a grandfather and his sister also had a tendency to bruise easily. She had had pleurisy in 1920 which had lasted for 4 months, during which time fluid had to be removed from the chest on one occasion. The cause of this was not known but tuberculosis was ruled out. She had had arthritis in both knees intermittently in recent years. There was a history of an old injury to the lower back from a fall at the age of 5 years, and a small granulomatous tumor had been removed from the right orbit in 1917 without recurrence or sequellae. She had had hepatitis at the age of 20 when she was ill for a month and recovered without complications. She had had pyelonephritis in 1958 with no recurrences. Her father had died at 74 of heart trouble; her mother at 58 of cancer; one sister died at 58 of a pulmonary embolus and one brother was living and well.

On examination, the temperature, pulse and respirations were normal. Her blood pressure was 170/98 but was 140/80 at time of discharge from the hospital. She was well developed, moderately obese, alert, ambulatory and in no distress. There were recent ecchymoses of the left arm, forearm, left leg and thigh. There was a large 5 x 12 cm. hematoma of the left iliac crest extending downward and posterolaterally. There were small 3 mm. purpuric spots inside the lower lip and a few in the buccal mucosa. There were remaining blood clots in the right nostril. Numerous petechiae were present in the skin of both lower legs. The general physical status otherwise was unremarkable except for Heberden's nodes of the fingers and irregular knee joint margins.

The hemoglobin was 15.0 Gm. (96%), the microhematocrit 43.5%, and the white cell count 6500 with a differential count of 63% polymorphonuclears, 17% lymphocytes, 9% monocytes and 1 eosinophil. The urine was acid, of specific gravity 1.016, and contained 5 mg % protein; no sugar or acetone was present and

the sediment was unremarkable. The prothrombin time was 14.5 sec., 70% activity, with the normal control of 12.6 sec. The BUN was 17.4 mg.%, the sedimentation rate 26.5 mm/hr., and the VDRL was negative. One stool gave a 1 plus reaction to guaiac. Blood platelet counts were as follows: 3/7/61: 92,000, 3/9/61: 98,000, 3/11/61: 120,000, 3/13/61: 180,000, and 3/15/61: 198,000. The clot retraction inhibition by quinidine was 64% compared with a normal control of 1.6%.

Her hospital course was quite uneventful. No new areas of purpura appeared and there was gradual resolution and absorption of the hematoma of the left hip area. She was advised as to her sensitivity to quinidine and cautioned never to take it again. There has been no recurrence of purpura in the year since this admission.

### DISCUSSION

It seems certain that this represents an instance of thrombocytopenia occurring as a hemotoxic effect of quinidine. Purpura appeared soon after the drug had been taken, disappeared after it was discontinued and was further documented by the positive clot retraction inhibition test. No attempt was made to verify it by other suitable *in vitro* tests<sup>1</sup> since the diagnosis seemed so certain and, in view of the warnings against use of an oral test dose of quinidine,<sup>2</sup> this provocative test under supervision was not employed.

Quinidine purpura has been known for more than 30 years but it has been increasingly recognized during the past decade when more frequent case reports have appeared in the literature. In a recent report of 6 cases, Bishop, Spencer and Bethell<sup>2</sup> estimated that their series brought to 47 the total reported up to that time. Of this total, there were 2 deaths attributed to quinidine purpura.<sup>3,4</sup> Females, particularly of the older age group, predominate over males in a nearly 5 to 1 ratio.<sup>5</sup> Many authors have considered purpura to be a rare complication of quinidine therapy when viewed in terms of the large number of patients taking this drug. To hematologists, however, it is not an uncommon cause of purpura. Bolton and Dameshek<sup>5</sup> have recently reported that quinidine now seems to be the most common drug causing thrombocytopenia.

The mechanism of development of thrombocytopenia in quinidine purpura has been adequately explained.<sup>1</sup> Quinidine attaches to the blood platelets in the sensitive individual to form an antigen which results in the production of a specific antibody. Action of the latter on the sensitized platelets results in agglutination, lysis and destruction with resultant thrombocytopenia and purpura. According to Dameshek,<sup>1</sup> quinidine and sedormid act similarly in this respect and they represent the first instances in which hemotoxic reaction to drugs have been clearly explained.

The diagnosis of purpura due to quinidine hypersensitivity depends upon its consideration as one of the

known causes of purpura. The history of recent intake of the drug is sufficient to incriminate it as a probable cause in any instance in which purpura occurs. Even when there is no history of intake of the drug, it can and should be ruled out by suitable in vitro tests. Once quinidine is incriminated as the cause of purpura in a hypersensitive person, the latter should be instructed never to take the drug again and to carry a memo card to this extent on her person. In almost all instances quinidine is rapidly removed from the body and the platelet count returns to normal within a week. Administration of adrenocorticoids or ACTH are probably unnecessary.<sup>2</sup>

#### SUMMARY

1. An instance of purpura due to quinidine in an elderly female who had been taking the drug intermittently for several months in treatment of episodes of paroxysmal auricular tachycardia, is described.

2. Survey of the literature reveals that purpura occurring as a toxic effect of this drug is not rare, that it

tends to occur mostly in elderly females, that it can be fatal and that the immunologic mechanism is known.

3. The diagnosis can be verified by suitable in vitro tests in preference to use of an oral test dose of the drug since the reaction to even a small test dose may be dangerous.

4. Spontaneous improvement follows discontinuance of the drug in most instances.

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237 Turner Street, Auburn, Maine

# Advantages And Practicality Of Owren's Thrombotest With Thoughts On Anticoagulation In General

W. S. GAMBLE, M.D.\*

#### INTRODUCTION

Owren<sup>2</sup> has developed a method of measuring the clotting factors suppressed by oral anticoagulants (eg. Dicumarol® and Warfarin). This represents a great advance in that other tests, although they measure some, do not measure all of the factors suppressed by anticoagulation. This test is reputed to be simpler than the Quick prothrombin time. It can be performed on capillary blood. It is not a bedside technique as inferred by the title of one article.<sup>3</sup> My purpose is to describe this test, its advantages and practicality, and to discuss its relationship to recent knowledge of clotting factors as influenced by oral anticoagulants. Also to be discussed is the rationale of anticoagulant therapy in acute myocardial infarction as related to these clotting factors.

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#### CLOTTING MECHANISM

A detailed discussion of the mechanism of coagulation is unnecessary. There are three independent systems which are concurrently in effect.

The vascular system relates to the integrity of the blood vessel wall.

The rapid extrinsic system requires the presence of tissue thromboplastin which, in the presence of various blood factors including factor IV (calcium), factor V (ac-globulin), factor VII (proconvertin) and factor X (Stuart-Prower factor) permits the conversion of prothrombin to thrombin. The latter promotes the conversion of fibrinogen to fibrin (the visible end-result of coagulation). In a normal individual this process is completed in the vicinity of 12 seconds.

The slow intrinsic system is usually initiated by contact with a foreign surface. This is called the contact factor, or Hageman factor. It may also be initiated by

thrombin formed by way of the extrinsic system. Thrombin acts on platelets, releasing a lipoid material, platelet (Co-) factor 3. The platelet factor (or factors) in the presence of various blood factors including factor IV (calcium), factor V (ac-globulin), factor VIII (antihemophiliac factor), factor IX (plasma thromboplastin component), and factor X (Stuart-Prower factor) triggers the formation of plasma thromboplastin which catalyzes the conversion of prothrombin to thrombin, the latter being necessary for the conversion of fibrinogen to fibrin. This process of fibrin formation, measured by the Lee and White coagulation time, takes about 12 minutes.

Of the above factors, only those which are affected by oral anticoagulants bear on this discussion. These are: factor II (prothrombin), factor VII (proconvertin), factor IX (PTC), and factor X (Stuart-Prower factor). These four factors are also those absorbed by barium sulfate. Factors II and X are involved in both systems. Factor VII is involved in the rapid extrinsic system. Factor IX is involved in the slow intrinsic system.

The one-stage Quick prothrombin time measures only factors II, VII, and X. Hence it completely reflects the defect in the extrinsic system induced by oral anticoagulants. It does not measure factor IX, and hence does not completely reflect the deficiency in the intrinsic system. The thrombotest of Owren measures factor IX, as well as factors II, VII, and X, and thus can detect any deficiency that can be induced in either the intrinsic or extrinsic system by oral anticoagulants.

PRINCIPLE OF OWREN'S THROMBOTEST

Owren devised an all-in-one reagent containing barium sulfate absorbed plasma which would reflect the combined degree of deficiency of the above four factors in the anticoagulated patient. The reagent also contains a tissue thromboplastin derived from a non-human species which prolongs the time required for the completion of the extrinsic phase of coagulation from 12 seconds (as measured by the Quick method) to 50 seconds. The reagent also contains cephalin which shortens the time for the completion of the intrinsic phase from 12 minutes to 50 seconds. Hence both systems can be measured simultaneously, allowing a detection of a deficiency of factor IX, which is involved only in the intrinsic system, in addition to the detection of deficiencies of factors II, VII and X. This reagent also contains calcium chloride.

TECHNIQUE

Basically, the technique consists of using 0.05 cc of blood or plasma mixed with 0.25 cc of the all-in-one reagent of 37°C. and recording the time of gel formation. Further details are given in other articles.<sup>2,3,4</sup>

NORMAL AND THERAPEUTIC RANGE

Normal thrombotest time ranges between 35 to 45

seconds or 70 to 130% activity. Percentage activity may be determined by a curve calculated for each batch of thrombotest reagent and included in the package from the company. Therapeutic range for activity is set as the same as prothrombin activity.

MATERIAL

Sixty-six tests\* were performed with the purpose of evaluating the technical aspects of the test. Normal and anticoagulated patients were studied to compare various modifications of the test. Five anticoagulated patients were tested on three consecutive days and three normal controls were taken at random. Tests were done on capillary blood and on venous whole blood and plasma. One stage Quick prothrombin times were measured simultaneously.

RESULTS

Some tests were invalidated by technical error and others, because of deliberate variables, did not lend their results for comparison. These results are not included.

Results on the three controls are listed in Table 1.

TABLE 1

RESULTS ON NORMAL CONTROLS				
	Thrombotest Venous Whole Blood	Plasma	Capillary Blood	Prothrombin Time
Pt. 1	45 sec	—	29 sec	13.5 sec
Pt. 2	41 sec	—	33 sec	14 sec
Pt. 3	44 sec	44 sec	40 sec	17.5 sec

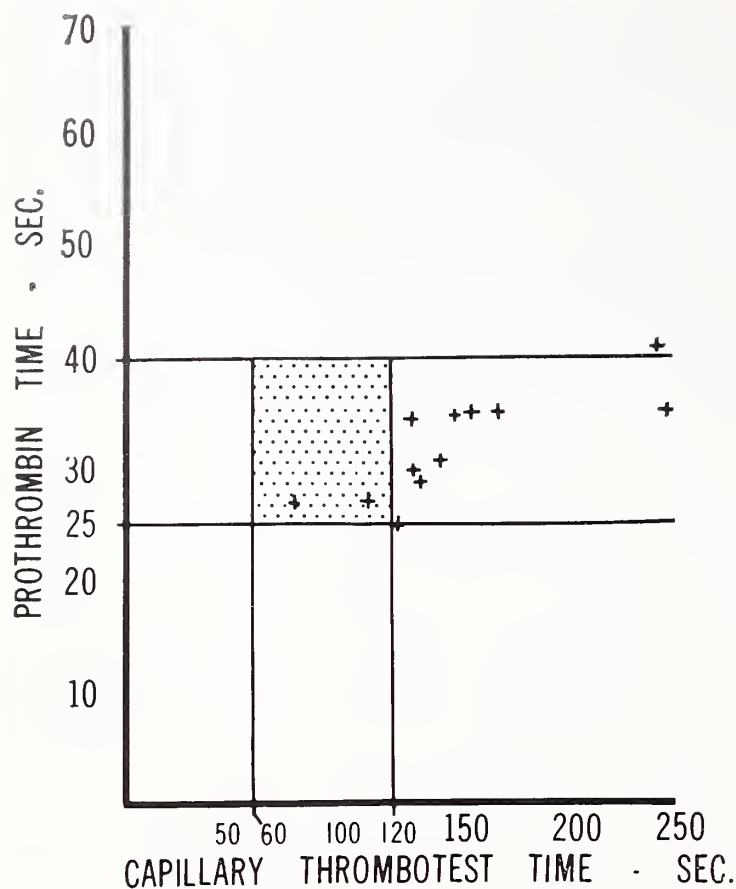
The capillary specimens correlate well with the prothrombin times. The results and relationship of prothrombin times and capillary thrombotest times are shown in two graphs. Graph 1 relates times to one another and Graph 2 relates % activity. The finding of generally lower thrombotest % activity than prothrombin activity has been observed by others.<sup>2,3,4</sup> This may simply reflect the additional depressed factor (IX) revealed by the thrombotest.

The values seen in the same patient on different days are interconnected by lines and labelled on Graph 3. There is reasonable expectation of correlation in direction of thrombotest times and prothrombin times from day to day in an individual patient. For two patients, correlation is fair. The other three correlate poorly; the arrows indicating the unexpected results.

The results showed no correlation between prothrombin time and venous whole blood or plasma thrombotest time.

The relationship of thrombotest times on plasma vs. capillary blood and venous whole blood vs. capillary blood are shown on Graph 4. There is fair correlation between plasma and capillary samples and between

\*Thrombotest reagent kindly supplied by Nyegaard & Co. A/S, Oslo, Norway.



GRAPH 1. Times (Sec.)

Therapeutic range represented by dotted area.  
See text for comment.

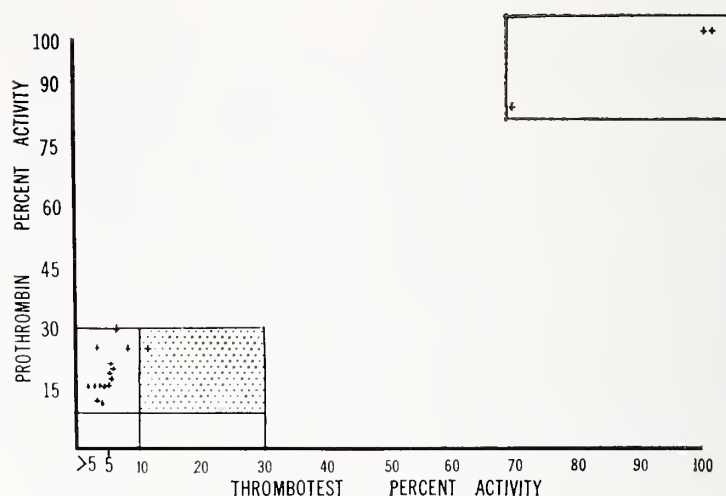
venous whole blood and capillary samples. The two minute and 15 second capillary thrombotest time (designated by arrows on graph) may have been in error in view of the good correlation of plasma and venous whole blood specimen taken at this time.

Venous whole blood and plasma were tested in three cases with shorter times resulting when plasma was used. Both plasma and capillary samples were tested in six cases with capillary samples giving shorter times in four cases.

One unexplainable result was obtained with the addition of calcium chloride solution as the solvent for the all-in-one reagent as recommended by Moore and Beeler<sup>4</sup> when using citrated blood. This was the prolongation of the thrombotest time in almost every instance. As noted above, calcium chloride is already a part of the thrombotest reagent. The amount, however, is not calculated to counter blood collected with added anticoagulant. One can circumvent this problem by using siliconized tubes without anticoagulant, and using distilled water for the solvent.

#### DISCUSSION

It is of little value to compare the thrombotest to Quick's one stage prothrombin time, because the prothrombin time cannot be taken as a standard. The prothrombin time itself is theoretically inadequate. Thrombotest % activity is not the same as prothrombin % activity, but this does not invalidate the



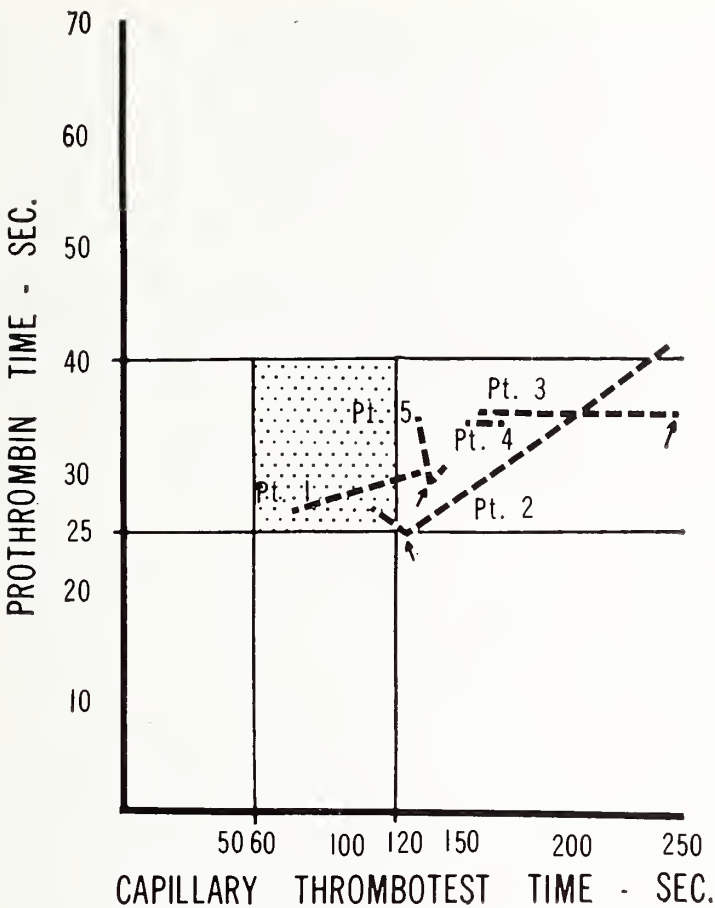
GRAPH 2. Percent Activities.

Therapeutic range represented by dotted area.  
Blocked off area at upper right designates normal controls.  
See text for comment.

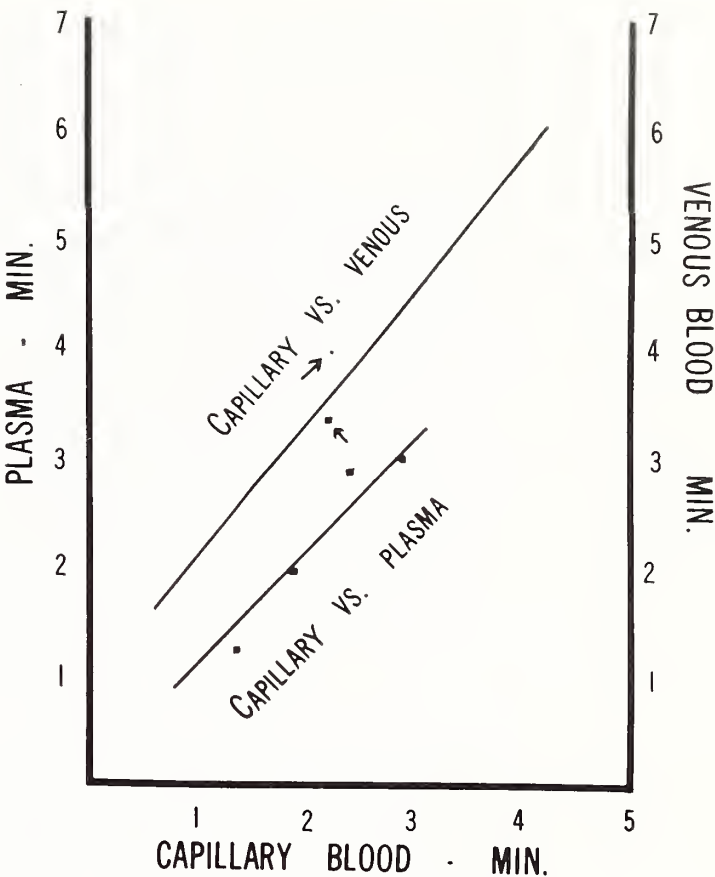
thrombotest. The only criterion would be to compare each to the standard which is the aim of both and that is the safe prevention of morbidity and mortality in thrombotic vascular disease. Only after a long term study of many patients could any conclusion be drawn between the two methods regarding hemorrhagic complications, occurrence of myocardial infarction, and fatalities from hemorrhage or myocardial infarction. Ideally required would be two groups paired for age, sex (including menstrual activity), weight, hypertension, and degree of disease. Both tests would be performed on each, but with one group controlled according to the thrombotest activity and the other group by prothrombin activity.

A clinical note of interest in this short trial was case #3. He developed purpura spontaneously on the first day and below the tourniquet on all three test days. Prothrombin activities were 12.5%, 16.5%, and 16.5%. His thrombotest activity was less than 5% on all three days. This was the only patient in the group who never exceeded 5%.

This test is simpler than the prothrombin time in that capillary blood can be used and the centrifuge can be eliminated. The time for clotting is longer with the thrombotest, and when multiplied by the large number of determinations done each day in some centers this disadvantage is magnified. Each test, at most, would take three minutes as activity is less than 5% at this point. The efficient performance of a number of tests simultaneously would minimize the above disadvantage. The technical skill required is less than with prothrombin time determinations in that an error of several seconds is of little significance when the normal is in the range of 50 seconds. The procedure is easily adaptable to office use. An office is more suitable than a hospital when the capillary method is used, as it is preferable to have the patient near a water bath. In the patient with inaccessible veins, the capillary approach is most helpful. How-



GRAPH 3. Fluctuations in Individual Patients. Therapeutic range represented by dotted area. See text for comment.



GRAPH 4. Thrombotest Times. Dots represent venous whole blood vs. capillary blood. Squares represent plasma vs. capillary blood. See text for comment.

ever, in this survey it was found that some patients preferred venipuncture to finger prick.

When performing the capillary method, free flow of blood is required. Milking the blood contaminates the specimen with tissue thromboplastin. A free flow is not difficult to establish when the prothrombin activity is below 30%. This becomes more difficult when the clotting mechanism is not suppressed. For collecting capillary blood a graduated 1 ml pipette works well when applied directly to the finger. The blood and reagent should be thoroughly mixed initially; but vigorous shaking is to be avoided subsequently as it will impede the formation of a clot. The end point should be looked for every five to 10 seconds after the first minute when testing anticoagulated patients. When using whole blood rather than plasma, recognition of the gel end point is more difficult. In either case, bubbles of air from shaking will obscure the end point. If whole blood is used, anemia will give lower times. The results should be appropriately corrected in this event.

The test may be used in measuring clotting defects in liver disease and is more comprehensive than the prothrombin time.

The cost of the thrombotest reagent for each determination is 12 cents vs. 6 cents for prothrombin reagent.

The large role anticoagulation has at the moment in

the physician's therapeutic armamentarium undergoes frequent critical appraisal by large controlled series. Its role in cerebrovascular disease is perhaps tenuous. Here, disease of limited extent, rarely associated with hemorrhage, characterized by recurring symptoms but often eventually fatal has received most attention. The role of anticoagulants has a better foothold in cardiovascular disease. The encouraging early reports of adequately controlled studies in coronary artery disease have brightened the outlook of a process associated with a high incidence, morbidity, and mortality. Cautionness was reflected by their use initially only after a second myocardial infarction. But the logic of this became elusive following demonstration of significantly reduced mortality. The tendency now is to use them earlier in the natural history of the disease, many introducing them in selected cases following the first signs of angina pectoris.

Most studies, however, have concerned themselves with their use in two phases of myocardial infarction, early and long term. Their early use to prevent thrombophlebitis, endocardial thrombosis, and thromboembolic complications has wide acceptance, although figures are presented occasionally to challenge this concept. An increase in myocardial rupture incidental to their use has been theorized, but whether this in fact is increased is controversial. If so, the net effect of anticoagulation still would seem to significantly

reduce immediate mortality. Their long term use is more controversial. Early studies showed anticoagulants to be valuable on a long term basis. More recent studies question this.

Studies of coronary artery disease have concerned themselves with what point in the disease anticoagulation should be used. Whereas in cerebral vascular disease early application is assumed, the main object becoming the more accurate definition of syndromes in the hope that results for specific situations may surpass those when cerebral vascular disease is taken as a whole. This difference in approach to cerebral and coronary vascular disease rests partly in the qualitative variation in functional defects of the brain, the area involved determining the functional disturbance. This disturbance may be relatively benign on the one hand, or fatal on the other hand. Although arrhythmias of varying prognosis depend on specifically localized myocardial injury, in general, cardiac reserve is more a quantitative matter.

In studies of elaborate planning, it is somewhat surprising that more concern has not been directed to the fact that the tests used for determining the amount of anticoagulant to be given do not measure all the clotting factors suppressed by the drug. The fallacy of this becomes very apparent when it is realized that inadequate suppression of these factors or excessive suppression may be overlooked if all clotting factors are not considered. The arbitrary therapeutic range of 10% to 30% prothrombin activity generally accepted, chosen presumably as the lowest, safest level of suppression, not only may not be the ideal range, but the occurrence of hemorrhagic complications as well as myocardial infarction within this range may have been predicted if all factors suppressed were measured. It would be unfortunate if the concept of anticoagulation was abandoned on the basis of studies which were based on measurements which failed to include all factors influenced by these drugs. This, then, is the main accomplishment of Owren's thrombotest.

I would like to comment on the timing and type of anticoagulant used in acute myocardial infarction. It is apparent that prothrombin time measures factors II, VII, and X. All of these are involved in the extrinsic system. Factors II and X are involved in the intrinsic system as well. The early maximal suppression of factor VII accounts for the early suppression of the prothrombin time within 48 hours. However, the maximal suppression of II and X does not occur until several (4 to 10) days following an oral anticoagulant, and factor IX, likewise, has a delayed response (4 to 6 days).<sup>1,2,3</sup> Because II, IX, and X are factors involved in the intrinsic system and this is the system of concern in intravascular thrombosis, it is apparent that adequate suppression with the oral anticoagulants

often does not occur until the fourth or fifth day despite the false assurance of the prothrombin time. Hence, if anticoagulation is desirable during the first five days following an acute myocardial infarction, heparin must be used concurrently. Thrombotest determinations are influenced by heparin. This must be taken into consideration if this test is done within eight hours of heparin administration.

It is my feeling, on the basis of the early occurrence of endocardial thrombosis, that heparin should be administered immediately along with oral anticoagulants in acute myocardial infarction if there is no contraindication to the use of these drugs. The heparin should be continued through the fifth day.

#### SUMMARY

The advantages and practicality of a new test for measuring clotting defects secondary to oral anticoagulants are discussed. The capillary method with thrombotest is simpler than the one stage Quick prothrombin time in several respects: simpler for the patient, less technical error, and avoids the need for a centrifuge. However, it requires two to two and one-half minutes longer to clot and requires proximity to a water bath. Efficient planning will minimize these disadvantages. If venipuncture is used, blood should be collected in siliconized bottles without added anticoagulant such as oxalate or citrate. When venipuncture is used, plasma avoids error in the presence of anemia and perhaps gives an end point easier to detect than whole blood.

The importance of this test lies in its measuring all factors depressed by oral anticoagulants in contrast to the prothrombin time. The significance of this, especially in the prevention of hemorrhagic episodes, but also in obtaining the ideal therapeutic range is apparent. Attempts at direct correlation to prothrombin time are not valid. It is urged that the only valid criterion is a large, well controlled study of sufficient duration relating the two methods to prevention of bleeding and prevention of morbidity and mortality from the vascular disease under long term treatment.

NOTE: I am grateful to Dr. Joseph Porter for his interest in the appraisal of this test, and to Dr. Irving Poliner for reviewing the manuscript.

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# The Journal of the Maine Medical Association

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## Across The Desk

### Mail Order Drugs

Mail order prescriptions represent a mushrooming problem in the quarters of day-to-day medical care and in the over-all field of public health. Checking prescription files is almost impossible with the mail order groups. It's small wonder that "drug reactions" are on the increase. In today's wonderful world when aspirin doesn't look like aspirin anymore, and "miracle" is a word used to describe a salad dressing, it's small wonder that the practicing physician views this new ogre with alarm. If Mr. Kefauver is sincerely interested in better medicines for the people, we respectfully suggest that this is one area where his talents would be both useful and appreciated.

### New? Rare Disease

A new rare familial disorder, characterized by enlarged odd-colored tonsils and low-blood cholesterol is providing scientists from the Public Health Service's National Institutes of Health with a unique opportunity to study previously inaccessible factors affecting the transport and storage of cholesterol and other lipids (fat-like substances) in the human body.

Called Tangier disease, because it was first discovered in two children on Tangier Island in Chesapeake Bay, the disease involves the accumulation of extremely large amounts of cholesterol esters in the tonsils and certain other tissues of the body.

The most striking biochemical feature of Tangier disease is the almost complete absence from the serum of high-density lipoproteins. These large molecules normally comprise one of the two major classes of fat-protein complexes that serve as carrier vehicles for all of the fats transported in blood. These lipoproteins have heretofore always been found in relatively stable

amounts in blood in man and all animals, and their specific function is unknown. They have received much less attention than the low-density of beta lipoprotein class, frequently linked to development of atherosclerosis.

The findings in Tangier disease suggest that high-density lipoproteins may be essential to normal handling of cholesterol, possibly including an important role in its normal esterification with fatty acids. (U. S. Department of Health, Education, and Welfare)

### Narcotics For Fall-Out Shelters

The question has recently been raised by well-meaning doctors as to whether or not it would be possible for them to stock-pile narcotics in their fall-out shelters for use after a possible atomic bomb attack. It has also been asked whether or not a doctor can write a prescription for narcotics for a trustworthy friend for stock-piling in his shelter. Although the intent of such conduct is no doubt for the very best, physicians should remember certain very important facts: First, it is unlawful for a physician to prescribe narcotics to a person other than a bona fide patient in the course of medical treatment. The physician would therefore be breaking the law if he were to write a prescription for a friend for the purpose mentioned. Second, it is unlawful for any person other than a doctor, or patient, or other specially authorized person, to even possess any narcotics. The kind friend to whom the physician supplied the narcotics would, also, be guilty of a violation of the narcotic law. Thus, both the physician and the friend would have to violate the present law in order to carry out their plan.

It would probably be possible for a physician, over an extended period of time, to acquire a stock of narcotics

which he could take with him into a shelter if the need arose. This could be done quite legally. There are certain practical problems in connection with this, however, which seem to require a deep consideration by the physician. It has been suggested by the physicians who have raised this whole question that narcotics, after an atomic attack, would be worth their weight in diamonds. This may be true. It is also true that a stock of narcotics is worth a lot of money today, even in the absence of an atomic attack. To place a supply of narcotics in any person's control, therefore, is placing that person in a position of danger to his family as well as to himself. If it ever became known, and such things do seem to become known, that Mr. X had a supply of narcotics in his fall-out shelter, every addict, peddler, hoodlum and racketeer would be making plans and attempts to get hold of the supply. That this would be so is illustrated by the number of instances in which attempts are made to steal narcotics from doctor's offices and automobiles.

Before a physician enters into a scheme like this he should realize, then, that he is breaking the law, that he is forcing his friend to break the law, and finally, that he is creating a very hazardous situation for himself and his friend and their families. (The Doctor & The Law — Vol. V, No. 2)

### **"Mediphone"**

A new national service for physicians, described as "the world's first center for emergency drug information," is scheduled to start dispensing information Jan. 8. This would be "Mediphone," a Washington-based operation offering the nation's physicians and their institutions a subscription service for speedy acquisition of latest information on new and old drugs. One of key backers is the NYC medical publicist, Dr. Cortez F. Enloe. Other directors include such known figures as California's Russel V. Lee, Ohio State Medical Dean Richard L. Meiling, NYC Nutritionist William H. Sebrell, Jr., and the Lovelace Foundation's Albert H. Schwichtenberg, all M.D.'s. (WRMS 755)

### **A Law Was Made**

*West Virginia Triumph* . . . The Kerr-Mills program in West Virginia — despite "premature obituaries" — is very much alive . . . On February 10, the State Legislature, after extending its off-year budget session for two days, approved a \$1.3 million appropriation for the program . . . With a matching federal contribution of \$3.08 million, this means West Virginia will have \$4,380,000 to operate the aged care program from July, 1962, to July, 1963 . . . Meanwhile, the so-called financial emergency which raised doubts about the current program proved to be exaggerated . . . A January 11 financial report showed that West Virginia still had \$4.3 million to expend on its Kerr-Mills until June 30,

1962 . . . At that time, the new appropriation will go into effect.

Following the February 10 action of the state legislature, D. E. Greeneltch, president of the West Virginia State Medical Association, issued a statement expressing the Association's appreciation to the legislators for continuing the Kerr-Mills program . . . At the same time, he noted that the \$1.3 million was only about half the amount appropriated previously for this program . . . Dr. Greeneltch said that "mature co-operation between the Department of Welfare and the various providers of service will be necessary" to make the program a success . . . Fees for services provided under Kerr-Mills, which had been cut back on December 1, 1961, have been restored . . . Eligibility requirements, which had been the most liberal in the nation, have been tightened. (Legislative Roundup, Feb. 16, 1962)

### **And This Is How It Is Done**

*AFL-CIO Campaign* . . . The AFL-CIO announced on February 13 that it will conduct a "crash" registration drive in an effort to elect an additional 30 union-backed House members to Congress this Fall . . . In explaining the need for this campaign, Al Barkan, deputy director of the AFL-CIO Committee on Political Education, said "we just don't have the votes" now to get union-sponsored legislation through Congress . . . Barkan added that the campaign will concentrate on precincts that were won by a narrow margin in 1960 . . . The AFL-CIO announcement emphasizes the importance of physician participation in the upcoming Congressional election campaign . . . As a Congressman-friend of medicine recently remarked: "You may be able to defeat the King-Anderson bill in the present Congress, but if you don't get out there and elect the candidates of your choice this year we may all become socialized by the next Congress." (Legislative Roundup, Feb. 16, 1962)

### **Eye Exam Detects Mild Measles**

A simple eye examination can detect a mild case of measles in which obvious symptoms are missing. Since mild measles provides immunity against the disease, the ability to diagnose such cases means that unnecessary preventive measures could be avoided.

A child given gamma globulin for protection against his first exposure to measles, for example, may not develop the fever, rash, spots or cough typical of measles and, therefore, be given more gamma globulin on later exposure. However, further gamma globulin injections could be eliminated if the eye exam confirmed that he suffered a mild case of measles.

Writing in the Feb. 17 *Journal of the American Medical Association*, Drs. Florman and Agatston said they had found that in both mild and regular measles a

slight inflammation of the cornea of the eyes and lining of the eyelids was present for a number of weeks. (News Release, AMA, Feb. 16, 1962)

### **Cervical Cancer Risk Higher In Early Marriage Group**

Women who marry in their teens face a higher probability of eventually developing cancer of the cervix, according to a report in the Feb. 17 *Journal of the American Medical Association*.

It was recommended that women who fall into this susceptible group seek regular gynecologic examinations. Cancer of the cervix can be cured when diagnosed in its early stage.

The Journal report was based on studies conducted at Kaiser Foundation Hospitals and Permanente Medical Group in Oakland, Calif., and San Francisco in which 155 cervical cancer patients were compared with 155 similar but cancer-free patients.

The study, in which all patients were questioned as to their earliest sexual experience regardless of marital status, revealed that significantly greater number of cancer patients reported such experience between the ages of 15 and 20 with cancer being diagnosed after a mean latent period of 30 years, I. D. Rotkin, Ph.D., Oakland, said. This relationship indicates the presence of some as yet unidentified cancer-causing agent, he said. (News Release, AMA, Feb. 16, 1962)

### **Medical Scholarships In Trouble**

The House Commerce Committee has rejected the President's plan to give federal scholarships to medical and dental students by a voice vote. The Committee will consider a substitute plan under which qualified students would be given federal loans rather than scholarships . . . The Committee has tentatively decided to expand the construction part of the program to give federal construction grants to schools of optometry, nursing, pharmacy and podiatry . . . Administration plan would have restricted such aid to schools of medicine, dentistry and public health. (AMA Council On Legislative Activities — Vol. 3, No. VII)

### **Blood Group Linked To Rheumatic Fever — Another Linked To Ca Of The Stomach**

A statistical association has been found between persons of a particular blood type and rheumatic fever, an article in the Feb. 17 *Journal of the American Medical Association* said recently.

Drs. Joseph A. Buckwalter and Gerald V. Tweed, Iowa City, Iowa, reported findings that indicated persons of NN blood type, a subtype of the MN blood group, had an "increased liability" to the disease.

Previous investigations had failed to turn up any

convincing evidence of an association of MN blood groups to disease, they said.

An increased incidence of rheumatic fever also was found among persons with one subtype ( $R^{2r}$ ) of the Rh blood group, they said.

The results of a four and one-half year study also justify the conclusion that there is an association between Rh blood groups and stomach cancer, the authors said.

The Rh blood-group effect in stomach cancer "seems to be a very strong one, since it is apparent in the relatively small group of 170 patients," they said. "However," they added, "the nature of the association is far from clear."

### **None Seem Eager To Face Medical Manpower Problem**

White House, Pentagon, Dept. of HEW and other Administration way stations are bemoaning a doctor scarcity, yet doing nothing to alleviate its effects. Selective Service's current efforts to produce 840 M.D.'s for military service are revealing bugs in tracedown machinery which are making large teaching hospitals howl that residency programs will be ruined. A "freeze" imposed by Dept. of Defense on PHS's medical self-help program could retard its advancement materially. While civil defense planning is being accelerated, its medical flotilla is rudderless. (WRMS No. 755)

### **Report Shows Numbers And Spread Of Elders**

Report being issued in Washington recently by the Population Reference Bureau, a private agency, highlights numerical and political importance of Americans 65 and over. They comprise 15 per cent of voting-age population and in past decade, while total population was growing by 18.5 per cent, they have increased by 34.7 per cent. Largest proportions of the aged are in New England and Plains states and smallest are in the South and Rocky Mountain states. Nationally they are 9.2 per cent of the population and ratios range from 5.4 in New Mexico to 11.9 in Iowa. In latter state, one out of every five voters is 65 or over. Before end of this decade it is estimated plus-65ers will rise to a strength of 20 million. (WRMS 766)

### **NIH Starts New Program To Check "Common Cold"**

Intensive study of respiratory infections is to be undertaken by a newly formed Vaccine Development Program at National Institutes of Health. Its head is Dr. Dorland J. Davis, associate director of Institute of Allergy and Infectious Diseases. An advisory board is chaired by Dr. Gordon Meikeljohn, Univ. of Colorado Medical Center. (WRMS 766)

# INTERIM MEETING

## Maine Medical Association House Of Delegates

Sunday, April 8, 1962

THE STOWE HOUSE, BRUNSWICK, MAINE

Dinner at 1:00 P.M. — Business Meeting at 2:00 P.M.

Presiding, Ralph C. Stuart, M.D., Guilford, President-Elect

### THE ORDER OF BUSINESS WILL INCLUDE —

Financial Statement for 1962

Proposed budget for 1963

Amendments to M.M.A. Constitution and By-Laws to provide for Speaker of the House as proposed by the following committee members: Drs. Linus J. Stitham, Robinson L. Bidwell and Arthur N. Lieberman. (This committee was appointed in accordance with a resolution approved by the 1961 House of Delegates and published in the September, 1961 issue of the Journal.)

Proposed amendments to the Constitution and By-Laws of the M.M.A.:

By-Laws Chapter IV: Add after Section 2, Section 2A as follows: A Speaker of the House of Delegates shall be elected at the annual meeting of the Association, by the House of Delegates, at a time to be designated by the Council and published in the agenda.

Section 2B: A Vice Speaker of the House of Delegates shall be elected in the same fashion as the Speaker.

By-Laws Chapter VI Section 2: Delete the third sentence reading "He shall act as presiding officer at all sessions of the House of Delegates and shall give a deciding vote in the case of a tie." Further delete the last paragraph reading "The President-Elect shall appoint a Committee on Parliamentary Procedure subject to approval

of the Council. Members of this Committee shall attend meetings of the House of Delegates and the General Assembly and other such meetings as requested by the President-Elect."

Add Section 4 reading The Speaker shall preside at meetings of the House of Delegates and perform such duties as custom and parliamentary usage require. He may address the House of Delegates at the opening meeting of all sessions limiting his address to matters of conduct and procedure in the House. He shall have the right to vote only in the case of a tie.

Add Section 5: The Vice Speaker shall officiate for the Speaker in the Speaker's absence or at his request. In case of death, resignation, or removal of the Speaker, the Vice Speaker shall officiate during the unexpired term.

Constitution Article VI Council: Add after the words "each Councilor District," and the Speaker of the House of Delegates.

Amendment to By-Laws Chapter VIII, Section 1 proposed by the Council: This amendment would change the deadline date for payment of State dues from April 1 to June 1.

Committee Reports

Complete agenda for this meeting will be mailed to each of the county delegates and alternates.

## Maine Medical Association Council

The Council will meet at the M.M.A. headquarters  
in Brunswick at 10:00 A.M.



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### Present And Projected Testing For Phenylketonuria

ELLA LANGER, M.D.\*

Following up on the excellent synopsis of "Some Current Studies in Phenylketonuria"<sup>1</sup> in the January, 1962 issue of The Journal of the Maine Medical Association, this discussion will present the preventive aspects of this disease — the approach in which this department is particularly interested.

At present only very few conditions leading to mental retardation are known as preventable if treatment is instituted early in life. One of these conditions is phenylketonuria.

It may be of general interest to know that 3 out of every 100 newborns are or will become mentally retarded. Studies have proved that an inborn error in metabolism i.e., a block in the metabolism of the essential amino acid phenylalanine results in severe mental deficiency. The disease manifests itself by progressive mental retardation, neurological symptoms — such as convulsions — and frequently eczema. Furthermore, the urine has a mouse-like, musty odor.

Since PKU occurs about once in every 20,000 births, it is estimated that there are probably 6000 to 9000 phenylketonuric mental defectives in the United States — about a fourth of them in institutions. It is important to know that mental retardation and other symptoms of the disease can be prevented if the condition is detected early, and treatment started as early as possible; or if mental retardation has become manifest, arrest of mental retardation can be hoped for. This disorder is inherited, caused by an abnormal recessive gene. It is hoped that genetic consultation on the inheritance of this disease, and detection of carriers among relatives of a diagnosed case, will become available in Maine in the future.

Treatment to arrest mental retardation or to improve mental development consists of a special diet low in phenylalanine. There are commercially available synthetic formulae which can be substituted for natural protein foods. One of these is Lofenalac, a casein

hydrolysate from which most of the phenylalanine has been removed, and carbohydrates, fat and vitamins added.

It is not possible to reverse the damage inflicted to the brain by the excessive phenylalanine in the body. However, in older children improvement in personality and behavior, accompanied by some improvement of their mental abilities has been reported. The excessive phenylalanine results from the failure to convert it into tyrosine.

The Division of Maternal and Child Health started a program for Mentally Retarded Pre-School Children in 1957, (from birth to 6 years of age) when a grant from the United States Children's Bureau became available to the State. At that time an evaluation clinic was initiated at the Thayer Hospital in Waterville, and at the same time a PKU screening program on a State-wide basis was begun. This is in line with screening programs for PKU now going on in many areas of the country. Many individual physicians and hospitals are also doing routine testing of infants. In Maine, the physicians conducting Child Health Conferences, agreed at the start of the Program to have the attending infants and pre-school children screened for PKU. The wet diaper test with ferric chloride for infants and a tube urine Ferric Chloride Test on older children is performed where possible.

In the clinic program three positive cases have been followed through treatment with low phenylalanine food. This product is being provided to the patient by the Division of Maternal and Child Health since the treatment is a long-term, expensive one and would place a big strain on the family if they were to provide this special food. The follow-up is being done in close cooperation with the research program now going forward at the Pineland Hospital and Training Center in Pownal. There, serum phenylalanine determination is provided once every three months or oftener when indicated. Psychological testing of these cases is performed at Pineland Hospital and Training Center about

\*Director, Division of Maternal and Child Health and Crippled Children's Services

three times a year and complete evaluation at the State Clinic for Mentally Retarded Pre-School Children at least once a year. This evaluation consists of examination by a pediatrician; psychological testing; nutrition and speech consultation and any other ancillary services for which need is indicated.

It may be of further interest to learn that a case detected at 6 months of age with an I.Q. of 54 has progressed within 6 months to an I.Q. of 80. No such spectacular improvement can be reported for the other two cases, one of which was started on the low phenylalanine diet at the age of 12 months and the other at 17 months. However, in both cases a definite improvement in behavior was recorded, although their I.Q. increased only slightly.

The Ferric Chloride Test is negative in the newborn. The reason is that the phenylalanine level has to be built up in the body before an excess will be found in the urine. This usually happens at 4 to 6 weeks of life. Therefore, the screening test with ferric chloride at the Child Health Conferences is performed at that age level if possible. The test is negative when plasma phenylalanine is about 20 mg or less per 100 ml plasma. (Normal concentration is about 2 mg per 100 ml.)

Recently a new test which proves positive during the first week of life has been described by Dr. Guthrie at the University of Buffalo School of Medicine.<sup>2</sup> This test, an inhibition assay, represents a new method which has proved positive in PKU cases still giving negative urine tests. This is described in the *Journal of American Medical Association*, No. 178, November 25, 1961.<sup>3</sup> The test can be applied for routine screening of newborns in the hospital. It is hoped that the new approach i.e., screening of newborns in the hospital by the blood phenylalanine-agar diffusion tests eventually will replace the screening of infants with ferric chloride; possibly in time, it will replace the screening urine test of older children. After the test proves positive it should be followed with serum phenylalanine determination.

It should be noted here that the Ferric Chloride Test performed with 5% to 10% ferric chloride in acidified urine sometimes gives false results especially when caused by intake of drugs, as for instance aspirin,

as well as some of the tranquilizers. As stated above, this test represents a screening method only, and positive and doubtful cases will need to have the quantitative serum phenylalanine determination. In Maine, these determinations are being done at the Biochemical Laboratory of Pineland Hospital and Training Center, Pownal, at the present time. Also, as mentioned above, laboratory controls of serum phenylalanine concentration have to be performed at that laboratory on all cases which have been placed on special diet.

Unlike the Ferric Chloride Test, the blood phenylalanine-agar diffusion test can be expected to give positive results within the first week of life. It is known that mental retardation has been prevented in cases which had been started on the special diet within the first weeks of life. Infants with this condition appear normal at birth. However, as shown above, the diagnosis can be made before clinical symptoms develop.

In summary, this represents an appeal to all physicians to consider the addition of the routine urine test for PKU to the examination of all infants in early life and to repeat negative tests once or twice during the first months of life. Efforts will be made as well to have the blood phenylalanine-agar diffusion test, a fairly simple, inexpensive test included in the complete examination of all newborns delivered in hospitals, prior to their discharge.

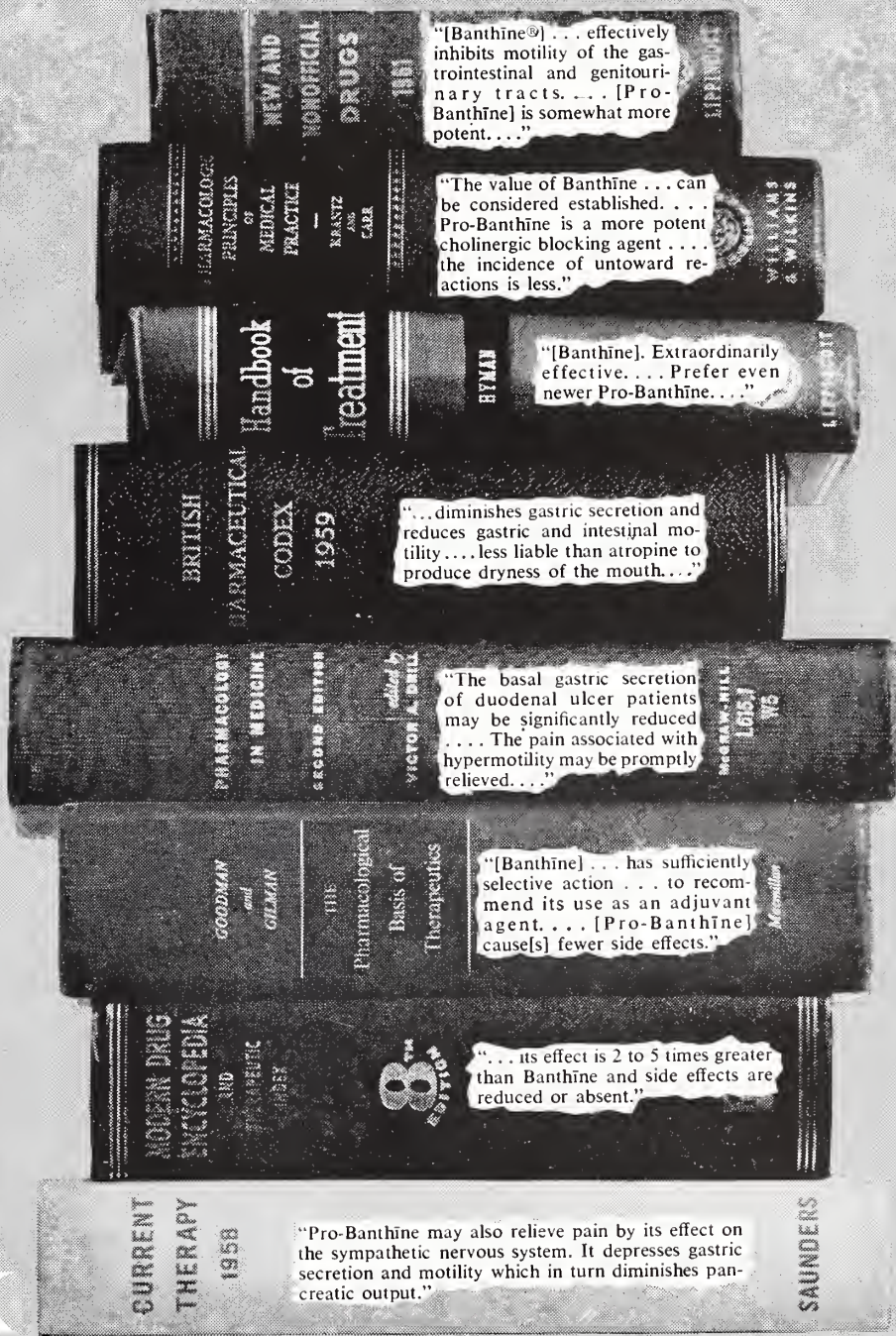
From the public health point of view, prevention of institutionalization presents a substantial economic saving, but more important, is the deep satisfaction of seeing a child — who otherwise may have needed institutional care — develop normally.

The Department has available on loan to physicians, hospitals, and allied groups in the State, a new film: "PKU — Preventable Mental Retardation" (15 min. — sound — color), which may be obtained upon request to the Department.

#### REFERENCES

1. Gruemer, H. D.: Synopsis of Some Current Studies in Phenylketonuria. *Journal of Me. Med. Assoc.* 8:15, 1962.
2. Guthrie, R. and Tieckelmann, H.: The Inhibition Assay. *Proceedings of London Conference on the Scientific Study of Mental Deficiency.* London, England, 1960.
3. Guthrie, R.: Blood Screening for Phenylketonuria. *Journal of Amer. Med. Assoc.* 863, 1961.

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# County Society Notes

## ANDROSCOGGIN

December 21, 1961

The annual meeting of the Androscoggin County Medical Association was held at St. Mary's General Hospital in Lewiston on December 21, 1961.

The financial report was given by Otis B. Tibbetts, M.D., Chairman of the Financial Committee. The society complimented the Chairman on the time, efforts and results of the endeavors of this committee.

The following officers were elected for 1962:

President, George B. O'Connell, M.D., Lewiston  
Vice-President, Morris Goldman, M.D., Lewiston  
Secretary-Treasurer, Donald L. Anderson, M.D., Lewiston  
Councilor, Cyprien L. Martel, Jr., M.D., Lewiston (3 yrs.)  
Delegate to the Maine Medical Association House of  
Delegates: Louis N. Fishman, M.D., Lewiston (3 yrs.).  
Alternates: Charles A. Hannigan, M.D. and Frederick  
B. Lidstone, M.D. of Auburn

Waldo A. Clapp, M.D. introduced the topic, "The Androscoggin Area Development Corporation" which was discussed by Drs. O'Connell, Harkins and Tibbetts and endorsed by the members present.

DONALD L. ANDERSON, M.D.  
*Secretary*

## HANCOCK

February 14, 1962

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on February 14, 1962.

Russell M. Lane, M.D., Vice-President, presided. The minutes of the previous meeting were read and approved.

Robert F. Russell, M.D. reviewed the recent proposal for Blue Shield coverage for those over 65 years of age. It was the consensus of our society that this proposed Blue Shield plan was being pushed upon the medical society under too much pressure and without adequate thought and discussion.

Thomas H. Palmer, Jr., M.D. of Bangor presented an interesting paper on Melanomas; a study with follow-up of cases at the Eastern Maine General Hospital from 1947-1960.

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## CUMBERLAND

February 15, 1962

A meeting of the Cumberland County Medical Society was held on February 15, 1962 at the Eastland Motor Hotel in Portland, Maine. Eighty-eight members were present.

Following a social hour and dinner, the meeting was called to order by the President, Robinson L. Bidwell, M.D. The obituaries of Roland B. Moore, M.D. and C. Earle Richardson, M.D. were read and it was voted that these be spread upon the records of the society and copies sent to the families.

The recommended amendments to the Constitution and By-Laws of the Maine Medical Association regarding "Speaker of the House" were presented by the President. These amendments are to be voted upon at the next meeting of the M.M.A. House of Delegates. After some discussion, it was voted to instruct the delegates to vote for the amendment creating a

speaker of the house, but to vote against that part of the amendments which make the speaker a member of the council of the Maine Medical Association.

Warren C. Baldwin, M.D. reported for the Grievance and Public Relations Committee giving a summary of the problems handled by his committee in the past two months. The Secretary reported for the Health Insurance Committee of the State Medical Association discussing the projected Blue Shield Program for senior citizens. Philip P. Thompson, Jr., M.D. discussed the use of funds provided by Kerr-Mills Bill in the State of Maine and this stimulated considerable discussion.

Upon question by George O. Chase, M.D. about the Recruitment and Careers Committee, it was announced that this committee had reorganized to include the Careers Committee of the Maine Medical Center Careers Sub-committee. The membership consists of Drs. Donald F. Marshall, Chairman, John F. Gibbons, Stephen E. Monaghan, Merle S. Bacastow, George O. Chase and Albert Aranson. Dr. Marshall announced that every high school in this area has been contacted regarding activities of this committee, and at a recent meeting of high school students at the Maine Medical Center, over a hundred students who were interested either in nursing or medical school attended.

ALBERT ARANSON, M.D.  
*Secretary*

## KENNEBEC

February 15, 1962

A meeting of the Kennebec County Medical Association was held on February 15, 1962 at the Senator Motel in Augusta with thirty-one members present.

The recent meeting of the Blue Cross-Blue Shield representatives with the committee on insurance was discussed, and a motion was made to approve the principle of a national Blue Shield program for elderly individuals. This was voted favorably by the majority.

Richard H. Dennis, M.D. of Waterville explained the indications and techniques of "corneal transplant" and stated that an Eye Bank in the area was necessary. It was voted to establish an Eye Bank at one of the Waterville hospitals and agreed that the Kennebec County Medical Association would sponsor the bank.

L. Edwin Sproul, Jr., M.D., a fellow in the Renal Unit of the Department of Medicine of the New England Medical Center, was the guest speaker of the evening. His talk on the use of kidney function tests in evaluation of renal disease was clearly illustrated with many slides and raised many questions from the floor.

EARLE M. DAVIS, M.D.  
*Secretary*

## PENOBSCOT

February 20, 1962

The monthly meeting of the Penobscot County Medical Society was held on February 20, 1962 at the Tarratine Club in Bangor, Maine. Thirty members were present with the President, Clement S. Dwyer, M.D., presiding.

Resolutions were read on the death of Henry C. Knowlton, M.D.

The society voted to endorse the Maine Medical Association.

tion's recommendations concerning insurance coverage for the aged.

It was voted to have a combined meeting with the Woman's Auxiliary in May. Preston A. McLean of Bangor was elected to membership in the society.

Mr. John McGinn, Trust Officer of the Merrill Trust Bank, guest speaker of the evening, spoke on the various means available to physicians to establish retirement funds. He discussed the ramifications of the Keogh Bill, systems allowing physicians to incorporate and the Uniform Gift to Minors Act. A discussion period followed.

FREDERICK C. EMERY, M.D.  
Secretary

## LINCOLN-SAGADAHOC

February 20, 1962

Fifteen members were present at the meeting of the Lincoln-Sagadahoc County Medical Society which was held at The Ledges in Wiscasset, Maine on February 20, 1962.

The Blue Shield Program for senior citizens was discussed and a motion that the county society approve this program was carried unanimously.

Anthony Betts, M.D. of Brunswick spoke on "Autoimmune Disease Reactions."

GEORGE W. BOSTWICK, M.D.  
Secretary

# News, Notes and Announcements

## University of Maine's Medical Services Club



Students at the University of Maine have organized a Medical Services Club for those in the pre-medical, pre-dental and medical technology programs. Through meetings and tours the Club encourages students to plan careers in the medical services. Officers are, left to right: president, Wayne L. Gerrish, Orono, senior majoring in zoology, pre-medical; treasurer, Johanna C. Hunt, Bangor, junior majoring in zoology; secretary, Gina Barnes, Newton Massachusetts, junior in zoology, pre-medical and vice president, Robin R. Loeschner, Bath, senior in zoology, pre-dental. Among those on the University of Maine staff working with the Club are Robert A. Graves, M.D., director of the University Health Service; Dr. Benjamin R. Speicher, head of the Department of Zoology and Mrs. Carol J. Hess, instructor in chemistry.

Moderator: Alfred F. Moseley, Harvard Medical School

### Participants:

Merritt Fitch, Sheriff, Hancock County  
"Origin of the Case and Background of Principals"

Richard C. Wadsworth, M.D., Pathologist Eastern Maine General Hospital  
"Nature of Skull Damage, Pathological Findings and Cause of Death, Identification of Body"

James H. Crowe, M.D., Medical Examiner, Hancock County  
"Cause and Manner of Death of this Victim"

Wilbur Ricker, Supervising State Fire Inspector  
"Origin and Spread of the Fires"

Kenneth Blaisdell, County Attorney, Hancock County  
"Test of the Polygraph and Prosecution of Accused"

Murray Burnstine, Automotive Engineer, Research on Fatal Highway Collisions, Harvard Medical School  
"Experiments"

Andrew Newcomb, Mechanic, Research on Fatal Highway Collisions, Harvard Medical School  
"Examination of the Car"

### NOTE

There has been a tremendous amount of effort put into the investigation of this case to be presented. This not only includes investigations conducted by Maine Officials, but scientific experiments conducted by Mr. Moseley's staff at Harvard Medical School. The program should be of extreme, important interest to the Medical Profession, Prosecutors and Law Enforcement Officials, all of whom are cordially invited.

## Department of Health and Welfare Division of Maternal and Child Health Including Services for Crippled Children (By Appointment Only)

### ORTHOPEDIC CLINICS

Augusta — Augusta General Hospital

1:00 p.m.: Apr. 26

Bangor — Eastern Maine General Hospital

1:00 p.m.: May 24

(Several will be two-session clinics)

Fort Kent — Peoples Benevolent Hospital

10:00 a.m.: May 9

Lewiston — Central Maine General Hospital

9:00 a.m.: Apr. 20, May 18, June 15

## Maine Medico-Legal Society Spring Meeting Augusta House, Augusta, Maine Wednesday, March 28, 1962

Social Hour: 5:30-6:30 p.m.

Dinner: 6:30 p.m.

### Program

Subject: *A Burned Body Found In An Abandoned Car*  
Co-operative Investigation Involving State, County Officials and Members of the Department of Legal Medicine, Harvard Medical School

Machias — Washington County Normal School

1:30 p.m.: Apr. 11

Portland — Maine Medical Center

9:00 a.m.: Apr. 9, May 14, June 11

Presque Isle — Arthur R. Gould Memorial Hospital

9:00 a.m. and 12:30 p.m.: May 8

Rockland — Knox County Hospital

1:30 p.m.: May 17

Rumford — Community Hospital

1:30 p.m.: June 20

Waterville — Thayer Hospital

1:30 p.m.: June 28

#### CARDIAC CLINICS

Bangor — Eastern Maine General Hospital

9:00 a.m.: Apr. 13, 27, May 11, 25, June 8, 22

Portland — Maine Medical Center

9:00 a.m.: Every Friday (Holidays Excepted)

#### CLEFT PALATE EVALUATION CLINICS

Portland — Maine Medical Center

10:00 a.m.: May 8

#### PEDIATRIC CLINICS

Bangor — Eastern Maine General Hospital

1:30 p.m.: Apr. 27, May 25, June 22

Presque Isle — Arthur R. Gould Memorial Hospital

1:30 p.m.: May 23

Waterville — Thayer Hospital

1:30 p.m.: Apr. 3, May 1, June 5

#### CLINICS FOR MENTALLY RETARDED

##### PRE-SCHOOL CHILDREN

Waterville — Thayer Hospital

9:00 a.m.: Apr. 4, 18, May 2, 16, 31, June 6, 20

#### ADOLESCENT CLINICS

Portland — Maine Medical Center

1:00 p.m.: Apr. 25, May 23, June 27

#### CYSTIC FIBROSIS CLINICS

(In conjunction with the Maine Medical Center, Portland)

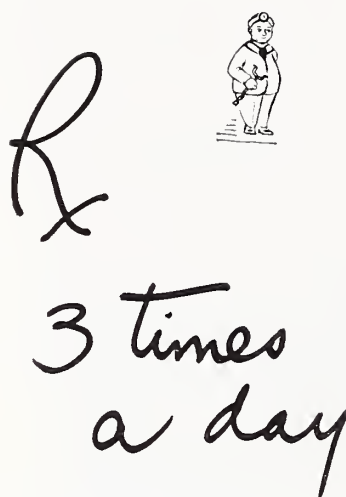
Portland — Maine Medical Center

9:00 a.m.: Apr. 17, May 15, 16, June 19

#### A COOKBOOK FOR MEDICAL EDUCATION

A cookbook of one hundred and fifty-six pages, containing recipes submitted by doctors' wives throughout the State of Maine and compiled by the Woman's Auxiliary to the Maine Medical Association. Every recipe in this book has been tested.

Copies of the cookbook will be available through the county society auxiliaries for a donation of \$1.25 — the proceeds to go to the Maine Medical Education Foundation. It is anticipated that this will mean approximately \$1,000 for this fund.



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# The Journal of the Maine Medical Association

Volume Fifty-Three

Brunswick, Maine, April, 1962

No. 4

## Pineland Hospital And Training Center Number

*This is the second issue of the Journal in honor of Hans V. Mautner, M.D.*



HANS V. MAUTNER, M.D., earned early recognition as research worker when he (and Adalbert Czerny) demonstrated the sphincter of the hepatic vein (1915) and when he explored pharmacological reactions of the sphincter in the following years.

As a teacher in the post-graduate program of the famed Viennese school from 1925 through 1938, he earned such distinction that the Soviet Union offered him the Directorship of the Leningrad Children's Hospital in the late 20's and his teacher, Czerny, in 1937, recommended him as Chairman for the Pediatric Department of the Karls University in Prague, Czechoslovakia.

Dr. Mautner, however, remained faithful to the Viennese faculty until the political turmoil of the 30's in Europe forced his emigration to the United States.

Here, he has served, with distinction, in several clinical and teaching positions, and since 1957, at Pineland Hospital and Training Center, where he became the first Clinical Director of the Center.

After World War II, Vienna twice (in 1946 and again in 1949) named him to become Chairman of the Pediatric Department of that famed university. Mautner declined the honor both times, for personal reasons.

Those who have shared, intimately, a part, or parts, of Professor Hans V. Mautner's professional life, have come to admire him for his integrity, his courage, his curiosity, and his knowledge. Beyond these rare virtues, Hans V. Mautner has earned, and commands, the affection of his friends and associates: as being a kind man of great wisdom.

# Etiology Of The Sturge-Weber Syndrome

## With Chromosome Analysis Of A Case

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In modern day medicine, with its emphasis on complex laboratory diagnosis, it is relatively rare that a rapid diagnosis is made on the basis of simple observation. The presence of a vascular nevus of the face in a patient presenting with convulsions is highly suggestive of the Sturge-Weber syndrome.<sup>1,2</sup> As Mautner<sup>3</sup> stated, tuberous sclerosis and Sturge-Weber's disease are the rare conditions in which the correct diagnosis of mentally retarded children is written on their faces.

Sturge-Weber's syndrome has appeared in the literature under a wide variety of names, including "encephalo-facial," "cerebrocutaneous," "encephalo-trigeminal" or "meningeal angiomatosis." Other names are "calcified pseudoangiomas of the brain," "benign sub-cortical calcifications" and "nevoid amentia." Although the names of Sturge,<sup>1</sup> Weber,<sup>2</sup> Dimitri,<sup>4</sup> Kalischer,<sup>5</sup> Krabbe,<sup>6</sup> have been linked eponymically with the syndrome, some of its features were first recorded by Schirmer<sup>7</sup> in 1860.

The first finding is usually a cutaneous angioma, in the form of a "port-wine" nevus in the distribution area of one or more of the major sensory trigeminal divisions, most often the ophthalmic branch. In almost two-thirds of the cases the nevus is present at birth. It is usually flat, but occasionally covered with rough nodules. Most often it is unilateral, tending to stop at the midline. Occasionally the entire face is covered, and more rarely the nevus extends to the trunk. The mucous membranes may be involved with cavernous nevi of the lips and the tongue.

Convulsions occur in over two-thirds of the cases, with onset from infancy to adulthood. They may be generalized or, more often, focal and contralateral to the facial lesion. The seizures may be difficult to control with anticonvulsants and surgical excision of the diseased cortex,<sup>8</sup> or even hemispherectomy, is occasionally carried out.<sup>9</sup> Contralateral transient or permanent hemiplegia or hemiparesis in about one-half of the cases usually is noted after the onset of seizures, but may precede the convulsive disorder. The neurologic evidence for weakness and spasticity may be rather subtle.

Mental retardation of variable severity is also found in about one-half of the cases. The eye is involved on the side of the nevus in about one-third of the cases, the findings varying from dilated vessels in the sclera and conjunctiva to glaucoma which may be secondary to angiomatous malformation in the choroid or uvea. Engorgement of retinal veins or actual retinal angiomas have been reported.<sup>10</sup>

Roentgenographic examination of the skull may reveal fairly characteristic sinuous calcifications arranged in double lines 1-2 mm apart. This finding is most often unilateral and on the same side as the facial nevus. It is most prominent in the occipital area and may be associated with skull asymmetry. Pneumoencephalograms may show atrophy of the involved side of the cortex with ventricular dilatation. The calcifications are not in the angioma of the meninges, but rather in the substance of the cortex below. Calcification has been reported as early as 4 months, but it may take a number of years before it becomes visible in roentgenograms. Angiograms occasionally demonstrate the angiomatous malformations of the meninges. Angiomas of viscera have been reported.<sup>11</sup> Hemiatrophy or hemihypertrophy on the side of the cutaneous nevus has been observed.

The Sturge-Weber syndrome in its complete form therefore includes: 1) cutaneous angioma, 2) seizures, 3) hemiparesis, 4) characteristic intracranial calcifications, 5) mental retardation, and 6) glaucoma. Many of the cases reported are incomplete and show only some of the above criteria. Several reviews on the subject are available.<sup>8,9,11,12,13a,b,c</sup>

For some time a group of syndromes involving the skin and the central nervous system have been collectively named "phacomatoses,"<sup>14</sup> "congenital ectodermoses,"<sup>15</sup> "neurocutaneous syndromes,"<sup>9</sup> etc. Conditions such as tuberous sclerosis, Recklinghausen's neurofibromatosis, Hippel-Lindau's disease, and recently ataxia telangiectasia have been covered by these terms. There have been suggestions to include the Sturge-Weber syndrome in this group of disorders which are admittedly genetically determined. Unlike the other conditions named, the etiology of the Sturge-Weber syndrome is still obscure. It has not been shown to have a simple hereditary or even familial pattern, although some authors believe in a more subtle mode of hereditary transmission.

According to Koch,<sup>16</sup> there exist reports of 19 cases in which the patient with the more or less developed

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syndrome had relatives with angiomatous nevi in areas supplied by the trigeminal nerve. In some cases, members of the kinship may have had seizures, migraine, mental retardation, or paralysis. Sorsby<sup>17</sup> stated that some reports suggested a "dominant form of inheritance but with considerable intrafamilial variability of expressivity." He further stated that he had encountered an affected mother and son, but he did not describe details of these cases. Louis-Bar<sup>18</sup> reported a young man and his maternal grandmother with a cutaneous angioma of identical localization on the face and right side of the neck as the only manifestations, which hardly can be called Sturge-Weber syndrome. Zellweger and Mikamo,<sup>19</sup> who found capillary hemangiomas in all 4 of the siblings and in several paternal uncles of their case of Sturge-Weber syndrome, believed that the condition is an example of dominant inheritance with low penetrance. Downing and Kreidberg<sup>20</sup> described a case of Sturge-Weber syndrome with 2 paternal cousins showing "birthmarks," one on the forehead, and the other on the posterior neck. Oppenheim's<sup>21</sup> cases of a 13 year old boy with pial angioma, hemiplegia, and Jacksonian fits, and his mother who had a cavernous angioma of the lip, may be cited here. In these families the relatives of the patients with Sturge-Weber syndrome had only very incomplete manifestations. It is true that the syndrome is a variable one and many cases are incomplete or "formes frustes." According to Blum and Mutrux,<sup>22</sup> 85% of the cases manifest only 1 or 2 of the cardinal symptoms which are said to be sufficiently characteristic to insure specificity. Furthermore, the complete picture may not be present at birth but develop with increasing age.<sup>16</sup> Therefore, an affected member of the family may die before the entire syndrome has developed and thus escape recognition. Such variability may cause a lack of pedigrees showing transmission of the disorder through several generations. In addition, even in cases of determination by a dominant gene, a syndrome or symptom may not be transmitted, if the first person to show the disorder is not capable or not likely to reproduce. Thus, a sporadic anomaly may be genetically determined.

If the pedigrees of patients with the Sturge-Weber syndrome showed such incomplete manifestations consistently, one could assume that a dominant gene with low expressivity was responsible for the symptoms or the entire syndrome. However, on the basis of isolated pedigrees containing such nonspecific symptoms as seizures, migraine, mental retardation, angiomas, etc., one cannot draw definite conclusions, since it appears possible that in such families pathologic manifestations of differing etiology have accumulated by chance. The pedigrees that have been referred to are selected and not convincing examples of determination by an incompletely dominant gene. It would also appear somewhat unusual for a disease with dominant inheritance to reveal no example of the complete syndrome being transmitted through more than one generation or

not being observed in more than one sibling (Louis-Bar,<sup>18</sup> Fraser,<sup>23</sup> McKusick,<sup>24</sup> Penrose,<sup>25</sup> Reed,<sup>26</sup> Refsum<sup>27</sup>). The only case of transmission of the syndrome, the case of Sorsby,<sup>17</sup> was not described in detail.

The patients of Geyelin and Penfield<sup>28</sup> reported as "cerebral calcification epilepsy" in a father and 4 of his children, were not cases of Sturge-Weber syndrome although they are cited at times as an example of the familial occurrence of the Sturge-Weber syndrome. The same can be said of a report of Touraine,<sup>29</sup> in which he described a pair of twins who had epileptic attacks and "voluminous varicose formation inscribed into the internal table of the parietal bones," but apparently no port-wine nevi. Since no illustrations were given of the patients or their roentgenograms, it is not possible to make a definite diagnosis or draw impartial conclusions from the report. In the older literature, reports of dominantly inherited Sturge-Weber syndrome were probably cases of Hippel-Lindau's disease.

Yakovlev and Guthrie<sup>15</sup> gave an interesting example of "a combined heredity" transmitted to a patient with the Sturge-Weber syndrome from both paternal and maternal sides. There were congenital ectodermal malformations, considered to be abortive forms of neurofibromatosis in the patient, in her mother, and in several brothers and sisters; a vascular nevus in 1 brother; fainting spells and convulsions in the mother and in several brothers and sisters. On the father's side there were nervous disorders, migraine and psychopathic disposition. In this family there was apparently a partial overlapping of Sturge-Weber syndrome with neurofibromatosis. The same was true in the case of Kissel and Arnould<sup>30</sup> in which the mother had neurofibromatosis and the daughter signs of Sturge-Weber syndrome. Possible transitions from Osler's hereditary hemorrhagic telangiectasia to Sturge-Weber syndrome were discussed by Chao.<sup>9</sup>

Recessive inheritance is also doubtful. It was suggested by Kirman<sup>31</sup> on the basis of "considerable cerebral morbidity on the father's side of the family," including congenital paralysis, convulsions, psychosis, hemiparesis or neurosis. How such findings can be interpreted as evidence of recessive inheritance is difficult to understand. A few case reports recorded parental consanguinity;<sup>16</sup> many others did not. It is to be hoped that in future publications attention will be paid to this part of the family history.

Other possibilities suggested by Penrose<sup>25</sup> were "fresh mutations of a gene presumably dominant," "intrauterine environmental accidents at a very early state in development," "somatic mutation of an unstable gene," and "infective disease of the fetus." Spiller<sup>32</sup> reported a case of Sturge-Weber syndrome in which a history was obtained of the mother having been exposed to roentgen rays of unstated dosage when she was 3½ months pregnant; the significance of this association certainly cannot be evaluated.

With lack of adequate genetic evidence to explain



FIG. 1. Face of patient showing on the left side the nevus in the area of the ophthalmic branch of the trigeminal nerve.

FIG. 2. Roentgenogram of skull showing in lateral view typical calcifications in the occipital area.

FIG. 3. Karyogram of cell derived from blood of patient showing normal 46/XX chromosomal constitution.

the etiology of the syndrome, the chromosomal study of Hayward and Bower<sup>33</sup> was encouraging in suggesting a possible cause. They reported trisomy of 1 of the 5 smallest acrocentric chromosomes, which they identified as probably chromosome 22. Following their publication, Lehmann and Forssman<sup>34</sup> reported normal idiograms in 2 cases of the Sturge-Weber syndrome. It was at this point that chromosomal studies were carried out in 1 of our patients, a girl with the Sturge-Weber syndrome. Subsequent reports by Hayward and Bower<sup>35</sup> in 7 cases, Hall<sup>36</sup> in 3 cases, Gustavson and Hook<sup>37</sup> in 2 cases, Zellweger and Mikamo<sup>19</sup> in 2 cases, Crawford and Ellis<sup>38</sup> in 2 cases, and McKusick<sup>21</sup> in 3 cases, described morphologically normal chromosome complements. It should be mentioned that recently Patau et al.<sup>39</sup> found a possible "partial trisomy" in 1 of their cases.

#### CASE REPORT

The patient observed by us, a 4 year old white girl, was the product of a normal full-term pregnancy and normal delivery. The mother was 27 years old when the child was born. She had had an operation on her "uterus and ovary" 2 months prior to this pregnancy. The father was 35 years old and allegedly in good health. There was no history of parental consanguinity. The patient's 9 year old sister had had 3 febrile seizures as well as breath-holding spells in infancy. A 14 year old brother was considered completely normal. The paternal great-grandmother, a maternal uncle, and possibly the paternal grandmother, had histories of seizures. The paternal grandfather, 2 paternal uncles, 2 paternal aunts, and possibly several paternal great uncles and aunts, were either blind or severely visually impaired; in some of these relatives a diagnosis of pigmentary degeneration of the retina had been definitely established.

At birth our patient appeared normal with the exception of a red birthmark noted around her left eye and in the left fronto-parietal area. At 3 months of

age, twitching of the right arm and leg was first noted. Neurological examination, lumbar puncture, subdural taps, skull roentgenograms, and electroencephalogram were all thought to be within normal limits. With the finding of the cutaneous angioma, a diagnosis of Sturge-Weber syndrome was considered.

Seizures continued to occur approximately every 3 months, lasting intermittently several days at a time. They consisted of turning of the eyes to the left, twitching of the right side of her lower lip, drooling, occasional minimal twitching of the right arm, and occasional urinary incontinence; however, there was no loss of consciousness or cyanosis. Transient weakness of the right arm and leg often followed these episodes. Phenobarbital, dilantin, sodium bromide, and mysoline have all been tried in various combinations without much success in changing the frequency of these episodes. Occasional strabismus has been noted. She was slightly hyperactive and demanding, and temper tantrums have occurred. Developmental history, including motor, language and social development, was considered within normal limits.

Physical examination revealed an alert, cooperative 4 year old white female. Height and weight were within the tenth percentiles. Head circumference was 47.5 cm. (close to two standard deviations below the mean for age). A port-wine angioma was noticeable about the left eye extending onto the left side of the forehead and down the left side of the nose (Fig. 1). There was a café au lait spot on the right buttock. The conjunctiva of the left eye appeared injected and there was a slight ptosis of the left lid. An esotropia was present. The fundi were considered normal and there was no suggestion of increased intraocular pressure. A functional cardiac murmur was audible. There was slight flattening of the right naso-labial fold. Some twitching movements of the right corner of the mouth were noted. There was a questionable right plantar extensor response. She seemed to be left-hand-dominant.

No other abnormal physical or neurological findings could be detected at that time.

Urinalysis, electrocardiogram, and chest roentgenogram were within normal limits. The bone age (67 ossification center survey) was between 1 and 2 standard deviations below the mean for age. Electroencephalogram was interpreted as showing normal basic rhythm for age with suggestive evidence of random slow irregular waves in the left occipital and parietal fields. Roentgenographic studies of the skull showed hemihypoplasia of the left side of the cranial vault and the typical double row of curvilinear calcifications in the occipital region (Fig. 2). Buccal smear for sex chromatin masses was in the female range.

Psychological testing using the Stanford-Binet scale (Form L-M) at chronological age 4-3 showed a mental age of 2-11 with an I.Q. of 67. Scatter was from year 2 to year 3-6. On the Peabody Picture Vocabulary Test she attained a mental age of 2-3. Her range on the Merrill Palmer Scale was from 24 to 47 months. It was felt that she had moderate mental retardation. In addition, she demonstrated problems in visual perception and seemed somewhat emotionally immature.

Skin biopsies were taken from the right and left forearms at the Children's Hospital in Cincinnati, placed in Eagle's basal medium supplemented with 10% bovine serum, and sent by air express to Dr. Ernest H. Y. Chu, Oak Ridge National Laboratory, where the biopsy specimens were cultured. The cell culture and cytological techniques have been previously described.<sup>40</sup> Cytological preparations were also made in Cincinnati from short term cultures of peripheral blood, utilizing the method of Moorhead et al.<sup>41</sup> Seventeen cells from the left skin biopsy and 11 cells from the right were studied by Dr. Chu, all showing 46 chromosomes including 2 X chromosomes. The karyotype appeared normal. Of 11 cells derived from leucocytes, 10 were 46/XX in type (Fig. 3), and 1 cell had 45 chromosomes.

#### DISCUSSION

This child fulfilled the requirements for a clinical diagnosis of the Sturge-Weber syndrome. She had a port-wine angioma in the distribution area of the ophthalmic branch of the trigeminal nerve, contralateral focal seizures with transient hemiparesis, a mild to moderate degree of mental retardation, and the typical roentgenographic picture of the curvilinear calcification in the occipital cortex ipsilateral to the facial lesion. There was no evidence of glaucoma.

The finding of 46 chromosomes with a normal idiogram is consistent with the normal chromosomal findings in the 21 other cases reported, and is, of course, at variance with the single case report of Hayward and Bower.<sup>33</sup> It is only fair to state that the latter authors in their original publication wisely avoided the assumption of a direct causal relationship between the trisomic condition and the phenotype of the syndrome, and suggested that the findings may well be a chance association.

#### SUMMARY

A case of a 4 year old white female, fulfilling the clinical criteria for a diagnosis of the Sturge-Weber syndrome is reported. Chromosomal studies of the cultured leukocytes from circulating blood and of cells of the skin grown in tissue culture revealed a normal 46/XX chromosomal constitution. The literature on the etiology of this syndrome was reviewed and it seems that no evidence for a definite chromosomal or genetic causation has been established.

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# The Clinical Aspects Of Accelerography\*

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## INTRODUCTION

With the growing urge to expand our knowledge on the mechanical performance of the heart, increasing attention has lately been paid to cardiac vibratory mechanics. Heart beat and blood flow result in mechanical vibrations rhythmically extending all over the body. It is customary to refer to rhythmic movements of limited body areas (radial, or femoral pulse, apical thrust, etc.) as *pulsations* while pulsatory vibrations of the whole body are identified as *ballistic* movements. The ballistocardiogram represents motions of the body as a whole including limbs, head, and abdominal organs. Precordial pulse tracings reflect vibrations of the thoracic surface in the close vicinity of the heart.

Some of the pulsatory (and ballistic) motions are in or near the range of the basic heart rate (1-2/sec.). Other vibrations are more rapid (from 30 to 1000-2000 per second). The number of vibrations per second determines the oscillatory frequency range (cycles/sec.). Generally, ultra-low and low frequency vibrations are sensed as motions, while high frequency oscillations belong to the audio range. The spectrum of body vibrations — including acoustic, pulsatory, and ballistic elements — consists of a mixture of low, medium and high frequency vibrations. No single instrument in use can record the entire vibratory spectrum. Special instruments, microphones, filters, and amplifiers have been devised to study heart sounds. Other devices serve to register the low frequency elements of pulsatory vibrations.

The movement of a body can be described by referring to the traveled distance (displacement) but this is not sufficient as information is also needed in regard to the duration of the movement. This can be supplied by devices representing the movement as a function of time (displacement tracing). Other devices supply information on the time course of the *velocity* of the movement. Velocity is a mathematical derivative of displacement. Further, one can study acceleration, the second mathematical derivative of displacement; acceleration can be defined as the rate of increase of velocity.

The acceleration of thoracic vibrations was first studied by G. Landes (1940). A seismograph was placed over the chest and its output was registered. The vibrations were synchronous with the heart beat. The pattern of vibrations of the acceleration tracing depended on the position of the seismograph on the chest.

Dorsoventral oscillations resembled a low frequency phonocardiogram, while those spreading over the thoracic surface in a head-foot direction, were similar to a ballistocardiogram. These classic experiments laid the foundations for the vectorial concept of cardiac vibratory mechanics. A firm mathematical — physical relationship was recognized between pulsatory phenomena and heart sounds, and between head — foot and dorsoventral ballistic vibrations.

Displacement, velocity, and acceleration are 3 different aspects of the same movement. Multichannel recorders and electronic computers are available for a simultaneous record of these three parameters of the movement.

U. S. and foreign research groups apply a variety of techniques to study longitudinal and vertical body vibrations. These studies have one common goal: the collection of data on the vibratory energy created by the heart in health and disease. It is not the objective of this paper to discuss theoretical or technical differences between various methods. Values common to these outstanding pioneer studies, rather than dividing factors, should be stressed in a trend for standardized procedures.

Standardization should be easily performed by simultaneous records of displacement, and acceleration or velocity, in a defined frequency band (probably 2-30 c.p.s.) and in standardized (head-foot or vertical) planes. The task of obtaining standardized tracings should not be difficult. Actually, there are but small differences between the various types of vibrograms when physically well defined recording techniques and frequency bands are used (Rosa and Luisada, 1959).

A series of recent projects has focused attention on *abnormal vibratory patterns in coronary heart disease*. The ballistocardiogram is abnormal in a high proportion of patients with coronary heart disease (Starr, Scarborough, Davis, Moss, Penneys and others). Agress and associates suggest that the vibrocardiograph combined with hypoxia and exercise tests may offer an even more sensitive method for the detection of coronary insufficiency. Kinetocardiography, a thoroughly studied technique to record ultra-low frequency tracings of precordial displacement, proved to yield valuable information on coronary patients (Harrison, Suh, Eddleman, Sheldon Skinner and others). Though neither of the above methods permits quantitative determination of cardiokinetic forces, statistical data of independent groups on the incidence of graphic abnormalities are surprisingly consistent. All available evidence points to

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a pronounced significance of vibratory abnormalities in ischemic heart disease.

PRECORDIAL ACCELEROGRAPHY

To date, accelerometers are simpler (Rosa; Elliott; Packard and Kyrazis; Mounsey; Hollis) than those used by Landes. The required supplementary equipment consists of a two-channel electrocardiograph. One channel is used to record the accelerogram while the other records an electrocardiogram for time reference. The precordial acceleration tracing (PACT) is a resultant of numerous vectors. It represents peak values of pulsatory acceleration in the dorsoventral plane of the thorax. There is a linear correlation between the time course of pulsatory acceleration and the second time derivative of pressure changes within the great thoracic vessels (Rosa et al., Fox et al.). Similar correlations for velocity have been found by Harrison and Coghlan et al.

Details of the accelerographic technique have been described elsewhere. The evaluation of the tracing is based on relative amplitude, frequency, and timing of individual oscillations. The cardiac cycle is subdivided in 3 systolic and 3 diastolic phases. The number of abnormal phases can be used to evaluate the severity of the pulsatory disturbance. Classification grade O indicates that no abnormality is present, and all phases of the cardiac cycle are normal. Grade I identifies a borderline tracing. This is the case when the abnormality of the tracing is limited to one phase of the cardiac cycle. Abnormal tracings are labelled grade 2 to grade 6 according to the number of abnormal phases.

Major respiratory variations are considered abnormal. The incidence of diastolic abnormalities appears to be in a direct relationship with electrocardiographic and clinical signs of myocardial ischemia.

CLINICAL ASPECTS

It has been repeatedly observed (Starr, Scarborough and others) that abnormal ballistocardiograms are not infrequent among "normal" persons. This has been attributed to latent coronary disease or possibly other factors. This is an intriguing problem which has significant implications. Serious attention has been paid to this problem in accelerographic studies of this Laboratory. A possible answer is that of dividing "normal," "borderline," and "abnormal" tracings.

The incidence of *normal tracings* in the presence of verified coronary heart disease was found 1.5% (4 out of 260 cases).

By using the criteria of graded classification, *no abnormal tracings* have been found in normal subjects and non cardiac patients. Identical results were reached when other possible indications of a pulsatory disturbance were examined. Two of these criteria appear to be of special interest: (1) No diastolic graphic abnormalities occurred, and (2) no abrupt respiratory variations were present in tracings of normal subjects.

TABLE I

The incidence of diastolic abnormalities in the PACT in normal, hypertensive and coronary patients.

Group	No. of Cases	Incidence of diastolic abnormalities %
Normal	79	0
Patients suffering from noncardiovascular diseases	17	5
Arterial hypertension. Normal ECG	30	33.3
Arterial hypertension. Abnormal ECG	90	53.3
Anginal syndrome. Normal ECG	21	52.4
Anginal syndrome. Abnormal ECG	82	80
Anginal syndrome + arterial hypertension. Abnormal ECG	36	86.2
Anginal syndrome + chronic heart failure. Abnormal ECG	25	76
Old myocardial infarction	56	94.7
Possible recent infarction	22	90.9
Definite recent infarction	39	100
Total	497	

On the other hand, 66.6% of patients with episodes of precordial pain (angina pectoris) and 63.4% of hypertensive patients with a normal electrocardiogram had abnormal precordial tracings.

*Borderline Tracings.* Seventeen out of 17 non cardiac patients had borderline tracings. These patients suffered from predominantly abdominal and pulmonary diseases. Fourteen and six-tenths per cent of 82 patients with episodes of precordial pain having an abnormal electrocardiogram had borderline tracings. On the other hand, the ratio was only 2.5\* in 142 patients with severe coronary heart disease. Regardless of electrocardiographic findings, about 30% of 120 hypertensive patients had a borderline precordial tracing.

*The extent of the graphic abnormality.* Hypertensive overload and myocardial ischemia apparently interfere with vibratory mechanics. There is some parallelism between the extent of the graphic disturbance and the severity of the clinical picture. Most of the tracings (73.2%) taken from hypertensive patients were normal, borderline, or grade 2. These patients usually had no or mild complaints. The majority (69.3%) of patients with healed myocardial infarction had grade 3 or grade 4 tracings. Graphs taken from persons with a recent infarction were in their majority (71.2%) grade 4 to grade 6.

*Respiratory variations.* More than one-half of the tracings with abrupt respiratory variations belonged to severe coronary cases.

*Diastolic abnormalities.* The most significant graphic

\*This percentage rose to 4.5 if cases with non-verified infarct were also considered.

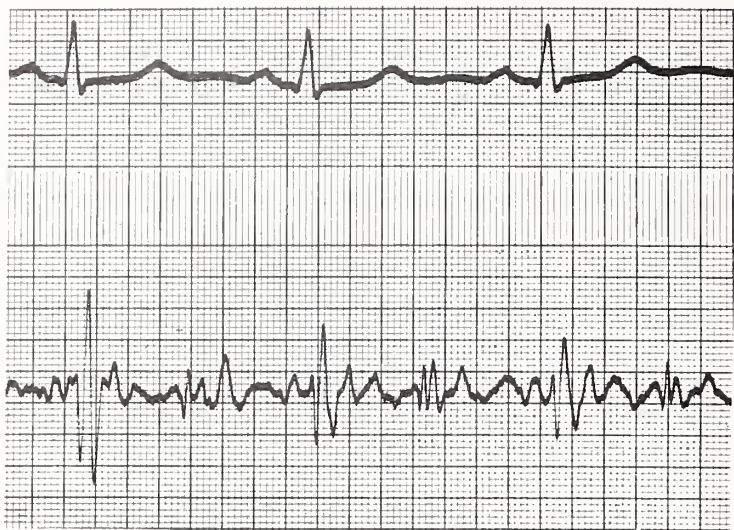


FIG. 1-A



FIG. 1-B

The effect of work test on the precordial acceleration tracing of a 55 year old male coronary suspect.

(A) before, (B) after exercise test.

Top tracings: ECG. Bottom tracings: PACT.

Film speed: 50 mm/sec.

(B) Note the overall increase in amplitude and the early systolic increase in frequency range. Large oscillations deteriorate the tracing in mid- and end-diastole.

criterion of myocardial ischemia or necrosis seems to be the deterioration of the tracing in diastole (Table I and Fig. 1). Such diastolic deterioration was never present in normal subjects. It has been observed in 5% of non cardiac hospital patients and in about 80% of patients with episodes of precordial pain having an abnormal ECG. The percentage rose to 100% in patients with a verified myocardial infarction. More than 90% of 78 patients with either an old myocardial infarction or acute coronary failure showed a deteriorated tracing in diastole.

Graphic abnormalities of the precordial tracing are reversible, and this may help in the evaluation of therapeutic measures. Active hypertensive medication, even bedrest, frequently result in the disappearance of one or

more abnormal patterns. Successful surgical correction of 230 cases of mitral stenosis (Rosa and Kunos, 1955) and 23 cases of patent ductus arteriosus (Rosa and Kunos, 1957) either restored the normal vibratory pattern or caused a considerable decrease in the number of accelerographic abnormalities. Such observations tend to prove that both changes of myocardial function and modifications of flow due to structural changes (valves, vessels) may be reflected in the vibratory pattern.

Accelerography is no specific diagnostic tool. No differentiation of hypertensive from coronary patients can be made by accelerographic criteria. Though some graphic data in valvular defects (mitral stenosis, aortic stenosis) seem to be characteristic, the number of observed cases has to be increased before drawing final conclusion. Tracings of patients with rheumatic heart disease and myocardial fibrosis plus reduced coronary blood supply may be similar to those found in coronary heart disease. These limitations do not detract from the value of the method in *screening* early or mild stages of cardiovascular disease and in evaluating the *extent* of functional disturbance in advanced stages.

In accordance with results obtained by other methods (Agress et al., Skinner et al., Scarborough et al., Penneys, Davis) we have found that an exertion test affects much less the cardiac vibratory pattern in normal subjects than in coronary patients. Observations in this Laboratory promise the possibility of a differentiation between various groups of "coronary" patients having similar electrocardiograms but obviously a different extent of myocardial damage (Fig. 1).

#### CONCLUSIONS

The authors have reviewed new methods which increase our knowledge of the mechanical performance of the heart. The possibilities and need for standardization of present methods dealing with cardiac vibratory mechanics are discussed. The use of a definite frequency band (possibly 2-30 c.p.s.), standardized pickups, and the simultaneous recording of more than one aspect (displacement and acceleration) of pulsatory — ballistic movements are advocated.

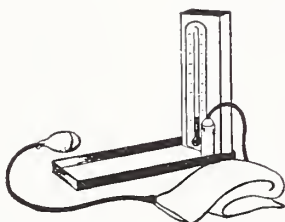
The accelerogram was studied in 497 clinically controlled subjects. No abnormal precordial accelerograms were encountered in normal cases. Only 1.5% *normal* tracings were found in subjects suffering from episodes of precordial pain and having an abnormal ECG, while 3 out of 4 had an *abnormal* PACT. The incidence of abnormal precordial tracings in hypertensive subjects, regardless of negative electrocardiographic findings, is 63-66%.

The number of graphically abnormal segments, particularly those in diastole, shows a linear correlation with increasing clinical and electrocardiographic evidence of myocardial ischemia. The incidence of such tracings in recent myocardial infarction was 100%. However, functional disturbances of the vibratory mech-

anism in hypertensive and coronary disease are not specific; abnormal tracings have been observed also in rheumatic heart disease with valvular lesions. Notwithstanding these limitations, the possibility of a distinction between normal and abnormal, and that of a quantitative evaluation of the vibratory disturbance, seem valuable as a supplement to conventional methods.

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# Symptomatology Of Unrecognized Chronic Brain Syndromes In Children\*

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## INTRODUCTION

In addition to the clinical pictures of mental retardation in its various forms and combinations described by Mautner<sup>1</sup> and others, it is plausible that relatively subclinical degrees of mental irregularity should exist, sometimes to be accompanied by minor motor abnormalities or cerebral dysrhythmias. Less obvious scattered patterns of irregularities on psychological testing and disordered behavior have long been recognized as a consequence of encephalitis.<sup>2</sup> The occurrence of hyperkinesis on an "organic" basis, frequently accompanied by abnormal electroencephalograms but without motor symptomatology, is a more recent concept,<sup>4,5</sup> Dijkstra<sup>6</sup> and his colleague, Precht<sup>1</sup> in the Netherlands have reported a frequent combination of minor choreiform movement of the fingers with difficulty in arithmetic and reading and lack of success at school. Other authors<sup>7,8</sup> have cast doubt on the existence of "a syndrome of minimal brain damage" and express concern lest it become established.

The purpose of the present paper is to outline a syndrome which with its variations is not very rare, but which seems to remain puzzling and difficult of explanation to parents, school teachers, and physicians. These children present for the most part borderline manifestations of chronic brain syndromes in 4 different areas of motor, intellectual, electroencephalographic, and perceptual deficits or irregularities. In most, 3 or more of the areas are involved, yet almost without exception the children had escaped recognition as having chronic brain syndromes.

## PRESENTATION OF CASES

Forty-one cases are presented which were seen in private consulting practice during a 6 year period. The condition is believed to be equally common in the neurological clinic of the out-patient department but since this is not a diagnosis coded according to the Standard Nomenclature of Diseases and Operations, there is no way of obtaining the records of this group nor of estimating its frequency.

The presenting complaint was almost always of poor school work, overactivity at home and at school, or of nervousness and emotional problems. The parents were often aware that the child had long been clumsy but motor disability (in the sense of excessive awkwardness) was the initial complaint in only 5 instances. Twenty-two were referred because of poor school work including poor handwriting, 7 for overactivity, 3 for nervousness or fearfulness, 3 for poor speech, and 1 because of an initial convulsion. The patients ranged from 4 to 12 years of age, plus 1 seen for the first time at 18. The 4 year olds had all attended nursery school or kindergarten and 29 of the 41 patients were in the age range 6 to 10 years. This would seem to indicate that exposure to educational pressures and appraisal by teachers are the most frequent way in which attention is directed to the children's difficulties. Some of the children had long been thought rather awkward but none had had a previous diagnosis of neurological abnormality made by a physician. It is to be noted, however, that 13 of 40 patients did not walk until after 18 months of age (the forty-first was an adopted child and the motor milestones were unknown).

The principal findings on neurological examination are presented in Table 1. The most frequent sign was mild choreiform twitching or athetotic posturing of the fingers of the extended arms, especially with the

TABLE I

Neurological Examination:	
Mild Choreoathetosis	16
Ataxia or Dysmetria	2
Tremor of hands	6
Hyperreflexia	14
Extensor Plantar Reflex	5
Spastic Paraparesis	1 (mild, previously unrecognized)
Clumsiness only	9
Normal except Behavior	1

TABLE II

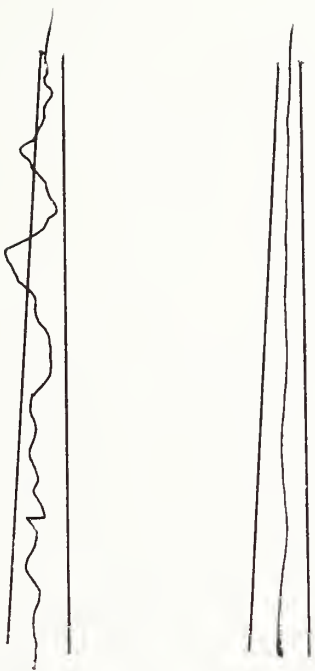
Speech:	Dysarthria	5
	Slow or mildly slurred	4
	Dysarticulation (sound substitution)	5
	Immature Quality for age	10
	Stutter	2
	Normal	22

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TABLE III

Mental Ability:	90-109	17	all at least mild "organic" irregularity
Formal I. Q.=110+		6	"
80-89		5	"
70-79		7	"
60-69		1	"
not tested		3	(but thought average)

FIG. 1  
At left, efforts of 9 year old patient to produce pencil trace staying between two converging lines. At right, normal control of same age.



eyes closed. Choreoathetosis in mild degree also often became evident in reaching for objects or in performing the finger-to-nose test, in which dysmetria or tremor of the hands were also sometimes revealed. Exaggerated tendon reflexes were present in fourteen, but positive Babinski signs were less frequent. One patient actually had an unequivocal but mild spastic cerebral paraparesis which had previously gone unrecognized. Nine patients showed no definite neurological abnormality except for excessive clumsiness, particularly with pencil and paper, but it is noteworthy that only a single patient was brought entirely normal except for the quality of his behavior. The quality of behavior, however, was often the most impressive feature of the examination. Overactivity, impulsiveness, distractibility, and short span of attention were soon evident. Many of the children were "office wreckers" in a way that seemed purposeless and almost unintentional. These characteristics of course improved with age but were striking when judged in comparison with other children of the patient's age at time of examination.

Speech, as outlined in Table II, was normal in about half of the patients but the others showed varying degrees of dysarthria, slowing or slurring, or of dysarticulation, plus 10 whose speech was evidently immature in quality for age and suggestive of mild mental backwardness rather than of a specific speech defect. It is of interest that of 34 patients whose hand dominance could be established with confidence, 13 were left

handed. In most of these, however, this was believed to be the result of a motor disability which was greater in the right hand, although relatively mild in either.

All but 3 of the patients had psychological investigations as given in Table III. A slight majority were of average or higher formal intelligence. All, however, showed at least mild irregularities of the "organic" pattern. These are detected only partially and only if of considerable severity by such standard tests as the Stanford Binet and the Wechsler Intelligence Scale for Children although relative difficulties in tests involving arithmetic are frequently striking. Better evidence is obtained by the use of form boards in the younger children and by such tests as the Kohs block designs and the Ellis design memory tests as well as others dependent on classification, generalization, abstraction, coding, etc. The poor perception of patterns and spatial relationships complicated learning to read and write and the situation was plausibly aggravated by awkwardness or tremor of the hands. Figures 1 and 2 show the effect of clumsiness, and of clumsiness in combination with cortical imperceptions. With increasing age, however, the children generally learned to read effectively, and spelling, which depends largely on rote memory, became the best school subject and arithmetic almost invariably the poorest. Discrepancies between the higher verbal and lower performance I. Q. scores were sometimes as great as 30 points. It was not uncommon for children to be able to perform arithmetic on only a second grade level, for example, but be able to keep up with fourth grade classmates in history

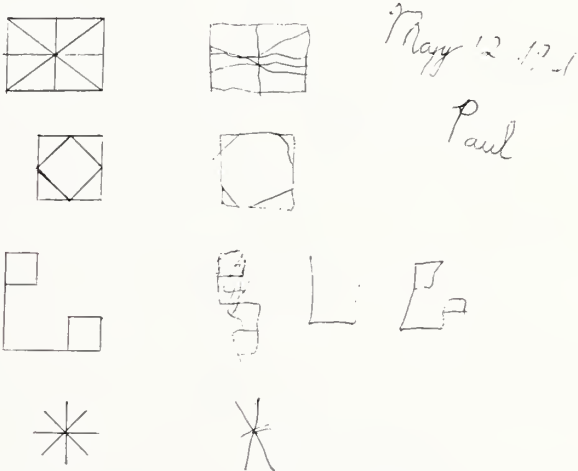


FIG. 2. At left, samples to be copied from memory, (shown one at a time from cards). At right, copies produced by 11 year old patient. Note one figure attempted 3 times. Upper right, patient wrote name and date.

TABLE IV

PREVIOUS POTENTIAL CEREBRAL INSULTS, BY HISTORY		
Bleeding in Mother's Pregnancy	2	
Long, hard labor (> 48 hours)	5	(followed by Caesarean section in 3)
Abnormal Presentation, difficult delivery	7	
Held back at Birth (15-45')	2	
Prematurity (B.W. < 5½ lbs.)	7	
Neonatal Complications		
Subdural Hematoma	1	
Cephalhematoma	3	
Convulsions	1	
Cyanotic Spells	1	
Postnatal Brain Insults		
Dehydration (severe)	1	
Meningitis	1	
Head Injury (severe)	1	
Entirely normal (1 unknown)	16	

and spelling. Difficulties in appropriate class placement are obvious, and the problem is compounded by behavioral characteristics which make the children disturbing influences in a classroom.

Electroencephalograms were made for only 17 patients but were abnormal or borderline in 15. The abnormalities were in voltages or rates only in 6 instances, but there were 5 with frank spike or spike-wave discharges. Interestingly enough, only 3 of the patients had had seizures. Two had had one grand mal fit each and 1 was subject to petit mal attacks.

As given in Table IV, the past medical histories of 16 of the patients and their mothers' prenatal and obstetrical histories were entirely free of what might be considered potential cerebral insults. The other 24, however (one was unknown), had histories suggestive of one or another abnormality which might be a plausible cerebral insult. While the interpretation of the etiologic influence of these factors requires a large scale prospective unbiased study, it is clear at once that the factors in the histories are very similar to those frequently obtained concerning patients in cerebral palsy clinics. It is of interest that Dijkstra's group of school children with minor chorea, short attention span, lability of mood and poor school work included 54% of a previous group of newborns with what he called the "hyper-excitability syndrome" of exaggerated jitteriness and myoclonic movements. Knobloch and Pasamanick<sup>9</sup> also call attention to possible minimal brain damage in a substantial percentage of premature as well as normal infants at age 40 weeks, and state a correlation factor of 0.75 with re-examinations at 3 years or later.

Mautner<sup>1</sup> has stated that roughly one-half of mental defectives of such degree as to require institutionalization come from families with positive family histories for mental retardation (some of such degree as the "Jukes" and "Kallikaks"). It is generally held, however, that low-grade mental defect in a child of intelligent parents is likely to be accidental (prenatal, natal, or postnatal) or the result of some rare recessive

genetic abnormality. The patients reported in the present study, it should be emphasized, were not the offspring of retarded or borderline parents. According to the widely utilized classification of the British Registrar-General, the parents of 16 were in Social Class I (professionals, proprietors of large businesses, higher army officers, etc.), 4 in Class II (semi-professionals, lesser managers), 6 in Class III (skilled workman, skilled clerical), only 1 in Class IV (semi-skilled) and none in Class V (unskilled). Similar conditions, however, are doubtless equally common in clinic populations of lower socioeconomic status, although different parental expectations may often render them less obvious, and less productive of emotional complications.

#### DISCUSSION

To present this group of children may appear to be belaboring the obvious, yet the fact remains that this syndrome (there is no intention to maintain it as a disease or diagnosis) is not uncommon and is usually unrecognized as such. It is difficult to interpret to the parents, schools, and physicians involved but doing so has frequently resulted in enormous easing of concerns all around. Emotional and behavior problems were extremely common. Twenty-one children were the subjects of major complaints of overactivity, tantrums, destructiveness, or disobedience, whereas 9 led to greater concern about their unhappiness, withdrawal, or fears. Only 15 children had no obvious emotional difficulty on examination and no parental complaint of any. In most cases the emotional problems abated to some extent with alteration of handling so that it is plausible to believe that they are to a considerable extent the reflection of conflicts between the child's characteristic thought processes and behavior and the environmental demands made upon him. Parents and teachers alike found it easier to make the required concessions when they came to understand that the discrepancy between a child's marks in arithmetic and in spelling was inevitable and not due to willful neglect or laziness. Even the distractibility and over-

TABLE V

<i>Area of Cerebral Function</i>	<i>Possible Overt Manifestations</i>	<i>"Borderline" Manifestations</i>
Motor	Cerebral Palsies	Minor Choreoathetosis or tremor Isolated Hyperreflexia Excessive Clumsiness
Mental	Mental Deficiency	Mild or Minimal Retardation Overactivity, impulsiveness, distract- ability, short attention span, low frustration tolerance, tantrums. Perseveration, concrete patterns of thought, difficulty in abstraction, dyscalculia
Sensory	Cortical Blindness or deafness Visual Field Defects Astereognosis, Impaired 2-point discrimination, etc.	Impaired memory for shapes, designs Impaired spatial concepts Visual or Tactile Inattention (Extinction)
Convulsive	Epilepsy	Abnormal EEG without seizures.

activity proved less disturbing in a classroom when its organic nature was understood and its victim permitted to be the boy designated to erase the black-board, open the windows, turn on the light, or similarly obtain a break from prolonged periods of (hopefully) sitting still. It is easy enough to conceive of similar possible areas for alterations in home management.

In this brief study no attempt has been made to evaluate approaches to treatment. Psychiatrists and child guidance counselors are frequently reluctant to undertake the psychotherapy of children with "organic" features. Admittedly these irregularities render therapy more difficult, but the need for it is no less great and frequently greater than among children of more even performance, so that it is to be hoped as, Lewis<sup>8</sup> has suggested, that this type of child should not be automatically excluded from therapy. No controlled or planned study of drug therapy has been conducted with the present group but the impression is similar to that of Laufer and Denhoff<sup>4</sup> that barbiturates frequently have a paradoxical exciting effect whereas dexedrine may equally paradoxically produce calmer and better organized behavior. Tranquillizers have in general proved disappointing. Anticonvulsant drugs have not been given to any of the present group not subject to seizures and it can only be stated that the question of treatment of the electroencephalogram (as opposed to the seizure) in the hope of improving behavior is a moot one.

SUMMARY AND CONCLUSIONS

Forty-one children are described who were referred for neurological consultation because of poor school work, overactivity, clumsiness, poor speech or emotional problems but without any previous definite diagnosis of neurological abnormality. Thirty-one showed definite abnormal neurological signs of the type demanded by some critics<sup>7</sup> of the "syndrome of minimal brain damage" although 9 of the other 10 were at least excessively clumsy. It is believed that legitimate

examples of the condition under discussion exist without motor deficit.<sup>10</sup>

Slightly more than half the patients were of average or higher intelligence but all showed disproportionate depression in some degree in tests involving design perception and memory, spatial orientation, abstraction, classification, in comparison with rote memory and verbal ability. Reading and writing were initially difficult and the latter complicated by awkwardness of the hands. In later primary grades, however, arithmetic was almost invariably the most difficult subject, particularly with respect to problems in which a manner of approach had to be selected, as opposed to memory items such as the multiplication table. The patients were also subject to overactivity, easy distractibility, impulsive behavior and short spans of attention as well as a low frustration tolerance which rendered their management at home and at school disproportionately difficult. Emotional problems were frequent but may have been to a large extent the result of the discrepancies between the nature of the patients' abilities and the demands made upon them.

Two of the patients had had one grand mal convulsion each and 1 was subject to petit mal but the others had no epileptic history. Nevertheless, electroencephalograms were abnormal or borderline in 15 of 17 for whom they were made.

It is proposed that there is a continuum of degrees and variations of impairments of cerebral function in 4 areas which may be stated broadly as motor, mental, sensory, and convulsive. The possible overt manifestations include cerebral palsies, mental deficiency, cortical sensory defects, and epilepsy but there are borderline manifestations in each area as indicated in Table V. The 41 patients presented probably all fall in the borderline region in each area in which they are involved. While all had escaped recognition as cerebrally abnormal, interpretation of the nature of the situation to parents and teachers is important for its understanding and acceptance, and for the child's emotional

well-being. While the basic irregularities can be changed little if at all, certain concessions in management are possible and may do much to improve functioning with the existing abilities.

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# Factors In The Pathogenesis Of Rheumatic Fever: A Study Of Streptococcal Infections And Rheumatic Fever Recurrences\*

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The purpose of this article is to present data which reveal some of the factors influencing the development of recurrences of rheumatic fever.

The data to be shown here were originally collected by the author during the 6 year period of September 1, 1939 to August 31, 1945 and were analyzed during 1948-50. Detailed results were presented on March 3, 1952 at a meeting of the New England Cardiovascular Society,<sup>1</sup> and portions of the data have been utilized for other papers by the author and his associates.<sup>2-10</sup> However, the only report of the entire study that has previously been published is a summary appearing in the Proceedings of the New England Cardiovascular Society.

Although a number of years has elapsed since completion of this study, several inquiries concerning our data received in the past few years and several recent reports of similar or related data by other investigators have indicated the desirability of publishing our findings in full. Furthermore, because our study was con-

ducted in the pre-penicillin era, it seems important to make our observations available for comparison with observations made since the widespread use of penicillin and other antibiotics.

In the present paper the data collected during 1939-45 are presented in 7 tables and 2 figures, which are the same as those presented before the New England Cardiovascular Society in 1952. No attempt is made here to discuss the significance of our data or to compare our findings with those that may have been published by others prior to or since 1952.

## CLINICAL MATERIAL AND METHODS

The clinical material utilized for this study is a group of approximately 900 rheumatic fever patients observed on the wards of the House of the Good Samaritan for a total of 410 patient-years during the 6 year period of September 1, 1939 to August 31, 1945. During this period blood specimens for determination of antistreptolysin-O titers were collected about every 10 days, and throat cultures for beta hemolytic streptococci were obtained routinely once or twice a week and at additional times whenever a patient developed fever or had a cold, sore throat, or other upper respiratory tract symptoms.

Throughout most of the 6 year period beta hemolytic streptococci isolated by culture were identified with regard to serologic group by the Lancefield precipitin technique and with regard to serologic type

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TABLE 1.

Annual Incidence of Streptococcal Infections\* and Rheumatic Fever Recurrences.

Year Beginning Sept. 1	Patient- Years	Streptococcal Infections*		Rheumatic Fever Recurrences	
		No.	Rate Per Patient- Year	No.	Rate Per Patient- Year
1939-40	81	77	0.95	36	0.44
1940-41	81	20	0.25	11	0.14
1941-42	71	14	0.20	3	0.04
1942-43	70	2	0.03	2	0.03
1943-44	56	14	0.25	4	0.07
1944-45	53	8	0.15	2	0.04
Five Years 1940-45	330	58	0.18	22	0.07
Six Years 1939-45	410	135	0.33	58	0.14

\*This analysis includes all clinical infections with a culture change or a definite increase in antistreptolysin-O titer and subclinical infections with an increase in antistreptolysin-O titer. Subclinical infections indicated by only a change in culture have been excluded.

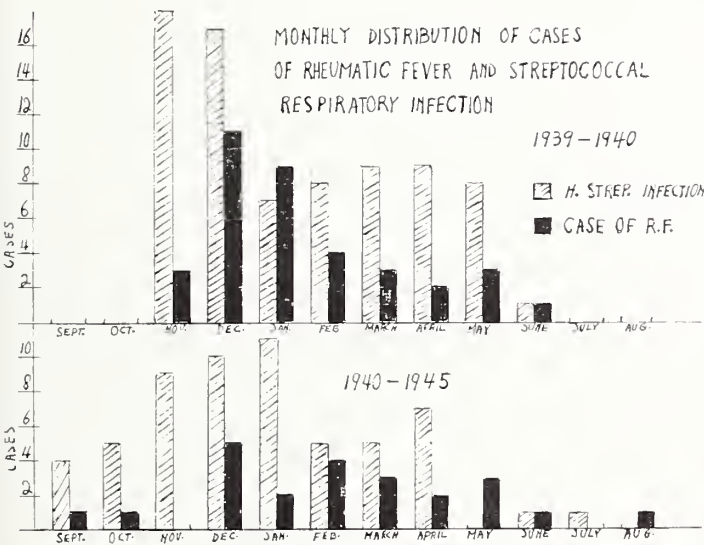


FIG. 1

by the Griffith slide agglutination method. The Lancefield precipitin technique for serologic typing of beta hemolytic streptococci was first used by us in March, 1944 but was not applied routinely until March, 1945. A patient was considered to have developed a streptococcal respiratory tract infection whenever there was a definite and consistent increase in the antistreptolysin-O titer by as much as 2 tubes or more or whenever there was a definite "change" in the throat culture findings. With regard to changes in throat culture findings, these generally consisted of an abrupt change from multiple negative cultures to strongly and consistently positive cultures for beta hemolytic streptococci. In some instances, when the patient was a streptococcal carrier, a change from a weakly and intermittently positive culture to a strongly and con-

sistently positive culture was accepted as bacteriologic evidence of streptococcal infection. In our hands the Griffith slide agglutination technique for typing streptococci was not found to be entirely reliable. When a patient who was a streptococcal carrier developed a change in culture, as defined in the preceding paragraph, there often was also a change in the Griffith type of the isolated organism. However, a change in Griffith type (in patients who were carriers) without a change in consistency of positive cultures or without an increase in heaviness of growth of streptococci on the blood agar culture plates could not be accepted as definite evidence of streptococcal infection. In a large majority of instances a definite increase in antistreptolysin-O titer was preceded by a definite change in culture as previously defined. On the other hand, a change in culture was not always followed by an increase in the antistreptolysin-O titer. Streptococcal infections, indicated by a change in throat culture findings or by an increase in the antistreptolysin-O titer, were classified as being clinical or subclinical, depending on whether or not there were symptoms or signs referable to the respiratory tract or fever.

RESULTS

*Incidence of Streptococcal Infections and Rheumatic Fever Recurrences.* In Table 1 the 6 years encompassed by this study have been divided into study-years, each year beginning on September 1. During the entire 6 year period there were 135 streptococcal infections indicated by (1) a clinical illness accompanied by a definite culture change with or without an increase in antistreptolysin-O titer or (2) by an increase in antistreptolysin-O titer without fever, sore throat, or other clinical manifestations. Nearly all of the subclinical infections with an increase in antistreptolysin-O titer were also characterized by a definite change in culture. Seventy-seven of the streptococcal infections (more than one-half of those included in this analysis) were observed during the year 1939-40. Table 1 also shows the distribution of 58 definite recurrences of rheumatic fever observed during the 6 year period. Not included here are 41 additional questionable recurrences. Thirty-six of the definite recurrences (nearly two-thirds of the total) occurred during the year 1939-40. Figure 1 shows the distribution of streptococcal infections and rheumatic fever recurrences by months for the year 1939-40 and for the 5 year period 1940-45. From the data in Table 1 and Figure 1 it is evident that in 1939-40 the incidence of these illnesses reached epidemic proportions. During the remaining 5 years both streptococcal infections and rheumatic fever recurrences occurred only sporadically. It is also evident that there is a rough correlation of the incidence of rheumatic fever and that of streptococcal infection. Both kinds of illness occurred most frequently during

TABLE 2.

Illnesses and Events Observed During 1939-45 and Their Relation to 58 Recurrences of Rheumatic Fever.

Event	Total No. of Events	Events Followed by Rheumatic Fever Recurrences.	
		No.	Proportion of Total Recurrences
Acute Tonsillitis or Pharyngitis	107	29	50.0%
Cervical Adenitis and Otitis Media	15	2	3.5%
Symptomatic Sore Throat	32	0	0%
Head Cold	101	1	1.7%
Bronchitis	13	0	0%
"Unexplained" Fever	102	13	22.4%
Miscellaneous Infections	51	0	0%
Tooth Extractions	174	0	0%
Subclinical Hem. Streptococcal Infections	85	13	22.4%
Total Rheumatic Recurrences		58	100 %

the winter and spring months and least frequently during the 4 month period of July through October.

*Clinical Events Preceding the Onset of Rheumatic Fever.* Table 2 shows the kind and number of illnesses or other events that were observed during the 6 year period of this study and their relation to the 58 definite recurrences of rheumatic fever. Fifty per cent of the recurrences were preceded by acute pharyngitis or tonsillitis, characterized by a painful and inflamed throat and fever\*. About 22% of the recurrences were preceded by fever (rectal temperature of 101° F or higher) unaccompanied by an inflamed throat or respiratory symptoms, and in an additional 22% of the rheumatic episodes there was laboratory evidence of an antecedent streptococcal infection without any fever, respiratory symptoms, or other manifestations of illness. Thus, in nearly 50% of instances the event responsible for the recurrence of rheumatic fever would, in all probability, have been overlooked had the patients not been under close observation in the hospital. These findings are consistent with and probably explain the common clinical experience that a history of an antecedent respiratory infection or other illness often cannot be obtained in patients first observed at the onset of initial attacks or recurrences of rheumatic fever.

The data in Table 2 also show that rheumatic fever episodes were preceded only rarely by an ordinary head cold and in no instance by symptomatic sore

\* At the time of this study in 1939-45 little attention was given to the presence or absence of exudate in the throat.

throat (painful throat without fever), bronchitis, or tooth extraction.

*Frequency with which Various Clinical Events Appeared to be Due to Streptococcal Infection.* Because of the relatively few kinds of clinical episodes that preceded the onset of rheumatic fever, it is of interest to consider what types of clinical illness are likely to be of streptococcal origin. Data bearing on this problem are presented in Table 3 and are summarized as follows: Acute pharyngitis or tonsillitis, which accounted for one-half of the recurrences of rheumatic fever, was frequently due to streptococcal infection. Unexplained fever, which accounted for almost an additional one-quarter of the cases of rheumatic fever, appeared to be of streptococcal origin in about 25% of instances. On the other hand, those events which usually were not followed by rheumatic fever (such as symptomatic sore throats, bronchitis and head colds) were very rarely accompanied by evidence of streptococcal infection.

*Relation of Rheumatic Fever Recurrences to Antecedent Streptococcal Infection.* Forty-six of the 58 rheumatic fever recurrences were preceded by definite bacteriologic evidence of a streptococcal infection. In 40 of the 46 infections there was a significant increase in the antistreptolysin-O titer as well as a definite change in the culture findings. In 10 recurrences there was highly suggestive but not conclusive bacteriologic evidence of an antecedent streptococcal infection, and 4 of these were associated with a definite increase in antistreptolysin-O titer. Thus, in only 2 recurrences was there complete absence of laboratory signs of streptococcal infection. These 2 recurrences were preceded by respiratory illnesses, but we could not demonstrate that the illnesses were of streptococcal origin.

Although nearly all of the 58 recurrences of rheumatic fever could be traced to antecedent hemolytic streptococcal respiratory infection, all streptococcal infections in rheumatic subjects were not followed by recurrences of rheumatic fever. In fact, of a total of 99 clinical illnesses with bacteriologic evidence of streptococcal infection only 35 (35%) resulted in new attacks of rheumatic fever, while of 85 subclinical streptococcal infections (shown by change in culture findings) only 11 (13%) seemed to precipitate rheumatic fever recurrences. Therefore, in the remainder of this discussion attention will be focused on the 184 illnesses or events in which there was bacteriologic evidence of hemolytic streptococcal infection, and an attempt will be made to evaluate some of the factors influencing the frequency with which such infections were followed by rheumatic fever.

*Relation of Clinical Characteristics of Streptococcal Infection and Antibody Response to the Development of Rheumatic Fever.* In Table 4 the 184 streptococcal infections have been divided into 4 categories according to whether or not they produced fever or other manifestations of illness (clinical or subclinical) and

TABLE 3.

Frequency With Which Various Clinical Events Appeared to be Due to Hemolytic Streptococcal Infection.							
Event	Total Number Studied	Bacteriological Evidence*		Rise in Antistreptolysin Titer		Bact. Evidence and/or Rise in Antistrep. Titer	
		No.	Proportion of Total	No.	Proportion of Total	No.	Proportion of Total
Acute Tonsillitis or Pharyngitis	107	64	60%	44	41%	69	64%
Cervical Adenitis and Otitis Media	15	8	53%	4	27%	8	53%
Symptomatic Sore Throat	32	1	3.1%	0	0%	1	3.1%
Head Cold	101	4	4.0%	2	2.0%	5	5.0%
Bronchitis	13	0	0%	0	0%	0	0%
"Unexplained" Fever	102	20	20%	17	17%	25	25%
Miscellaneous Infections	51	1	2%	2	4%	3	6.0%
Tooth Ex- tractions	174	1	0.6%	0	0%	1	0.6%

\*Definite "change" in throat culture findings.

TABLE 4.

Factors Influencing the Development of Rheumatic Fever Recurrences Following Definite Hemolytic Streptococcal Infections: <i>The Antibody Response and the Clinical Characteristics of the Respiratory Infection.</i>							
	With a Significant Rise in Antistreptolysin Titer			Without a Significant Rise in Antistreptolysin Titer			
	Total Infections*	Followed by Definite R. F. Recurrence		Total Infections*	Followed by Definite R. F. Recurrence		
Hemolytic Streptococcal Infections with Fever or Other Clinical Signs	56	31	55%	43	4	9.3%	
Subclinical Hemolytic Streptococcal Respiratory Infections	20	9	45%	65	2	3.0%	
TOTAL	76	40	53%	108	6	6.0%	

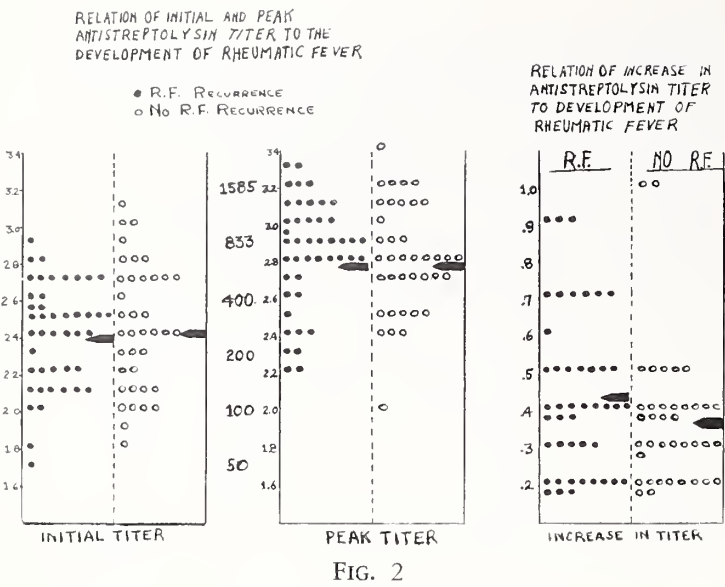
\*All streptococcal infections indicated by a change in the throat culture findings.

according to whether or not they resulted in a significant increase in the antistreptolysin-O titer. Also shown in Table 4 is the frequency with which infections in each of the 4 categories caused recurrences of rheumatic fever. The antibody response would appear to be an important determinant of whether or not a streptococcal infection precipitates a rheumatic fever recurrence. Thus, recurrences followed 53% of infections that caused a significant increase in antistreptolysin-O and only 6% of infections that were not accompanied by a detectable increase in the antistreptolysin-O titer.

Although the clinical characteristic of the streptococcal infection would also seem to play a role in its ability to produce rheumatic fever (clinical infections causing more recurrences than subclinical infections),

this factor would appear to be related to rheumatogenicity rather indirectly through its relation to antibody response. Thus, rheumatic fever recurrences followed 55% of all clinical streptococcal infections in contrast to 13% of all subclinical infections. However, when the analysis is restricted to infections with an antibody response, the difference in recurrence rates for clinical and subclinical streptococcal infections is very slight (55% as compared to 45%).

*The Initial Antistreptolysin-O Titer, The Peak Titer, and the Degree of Increase in Antistreptolysin-O as Factors in the Development of Rheumatic Fever Recurrences.* The relation of these factors to the development of rheumatic fever recurrences following streptococcal infection is shown by the data in Figure 2. In constructing this figure we utilized 76 episodes in which strep-



tococcal infection was indicated by both a definite change in the culture findings and a significant increase in the antistreptolysin-O titer. In addition, we included 6 infections in which a streptococcal origin was indicated by a significant increase in the antistreptolysin-O titer without accompanying bacteriologic evidence. An increase in titer was accepted as significant only when the increase was at least 2 tubes above the titer of serum collected at or shortly before the onset of the infection and only when the change in titer was confirmed by tests on several serum specimens.

From inspection of Figure 2 it is evident that the range of initial titers and the mean initial titer (shown by the arrow) as well as the range of peak titers and the mean peak titer were essentially the same for patients who failed to develop rheumatic fever as for those who developed recurrences.

With regard to the degree of increase in titer following the streptococcal infection, marked increases of 5 tubes or more were observed in 17 of 44 patients who had recurrences of rheumatic fever and in only 7 of 38 patients who did not develop recurrences. However, the interpretation of this possible difference is open to question since there was only a slight difference in the mean increases for the 2 groups of patients.

*Host Factors in Rheumatic Fever.* The host factors which influence susceptibility to rheumatic fever are poorly understood. One set of observations in the present study that may relate to this problem concerns the frequency of rheumatic fever recurrences in rheumatic subjects with rheumatic heart disease as compared to the frequency of recurrences in rheumatic subjects whose hearts remained undamaged. These data on recurrences, shown in Table 5, were obtained by analyses that were confined to patients whose streptococcal infections were followed by a significant increase in the antistreptolysin-O titer. Among 61 such patients with rheumatic heart disease (RHD) the recurrence rate was 60%; whereas among 15 similar patients with normal hearts (PRHD), the recurrence rate was only 20%.

TABLE 5.

Influence of Cardiac Status on Incidence of Rheumatic Fever Recurrences Following Streptococcal Infections. Analysis Restricted to Infections Followed by a Significant Rise in the Antistreptolysin Titer.

Cardiac Status*	Number of Infections Followed by a Significant Rise in Antistreptolysin Titer	Followed by R.F. Recurrences	
R. H. D.	61	37	61%
P. R. H. D.	15	3	20%
TOTAL	76	40	53%

\*RHD indicates rheumatic heart disease; PRHD indicates normal heart.

TABLE 6.

Annual Variations in Frequency of a Significant Antistreptolysin-O Response to Streptococcal Infection\*.

	Period	Total No. of Infections	Infections Followed by A Significant Increase In the Anti-streptolysin-O Titer	
Clinical Infections	1939-40	63	41	65%
	1940-45	36	15	42%
Subclinical Infections	1939-40	37	11	30%
	1940-45	48	9	19%

\*All streptococcal infection indicated by a change in the throat culture findings.

TABLE 7.

Annual Variations in Frequency of Rheumatic Recurrences Following Streptococcal Infection. Analysis Restricted to Infections Followed by a Significant Rise in Antistreptolysin and to Patients with Rheumatic Heart Disease.

Period	Total Number of Streptococcal Infections	Definite Rheumatic Fever Recurrences	
1939-40	41	29	71%
1940-45	20	8	40%
Total	61	37	61%

*Variations in Streptococcal Strains With Regard to Antibody Response and Rheumatogenicity.* As previously mentioned, streptococcal infections reached epidemic proportions during the year 1939-40 and occurred sporadically during the following 5 years. The epidemic strain of streptococcus, as compared to the various strains that caused sporadic infections, appeared to stimulate an antistreptolysin-O response more frequently and also seemed to cause a greater incidence of rheumatic fever recurrences.

Data relating to the antistreptolysin-O response are presented in Table 6. All infections (clinical and subclinical) utilized for this table were indicated by a definite change in the culture findings. Sixty-five per cent of clinical infections and 30% of subclinical in-

fections caused a significant increase in antibody titer in the year 1939-40 as contrasted to the corresponding figures of 42% and 19% for the 5 year period, 1940-45.

Table 7 presents data on rheumatic fever recurrence rates for patients with rheumatic heart disease in whom streptococcal infections were indicated by both bacteriologic signs (change in culture findings) and a significant increase in the antistreptolysin-O titer. Seventy-one per cent of 41 infections in 1939-40 were followed by rheumatic fever, whereas only 40% of 20 similar infections in 1940-45 resulted in rheumatic fever. Although these data suggest that the epidemic strain of streptococcus prevalent in 1939-40 was more rheumatogenic than the strains responsible for infection during the next 5 years, it should be pointed out that the numbers involved may be too small to attach significance to the observed differences.

#### SUMMARY

During the 6 year period from September 1, 1939 to August 31, 1945, 58 definite recurrences of rheumatic fever were observed among ward patients at the House of the Good Samaritan. All but 2 of these recurrences could be traced to antecedent hemolytic streptococcal respiratory infections. These antecedent streptococcal infections, in about 50% of instances, were characterized by typical manifestations of acute pharyngitis or tonsillitis. In the remaining cases the infection was either entirely subclinical or was accompanied by fever without symptoms and signs referable to the respiratory tract.

Although rheumatic fever in nearly all instances could be traced to streptococcal infection, streptococcal infections, even in known rheumatic subjects, did not always produce rheumatic fever. Therefore, an attempt was made to analyze those factors which played an important role in determining whether or not a streptococcal infection would be followed by rheumatic fever. Although the problem of the mechanism by which streptococcal infection sets off rheumatic fever remains unsolved, the following information of possible significance was obtained:

1. Streptococcal infections which stimulate an antibody response are much more likely to produce rheumatic fever than are infections which fail to alter the antibody level.
2. As long as there is a definite rise in the antibody level, the magnitude of the antibody response does not seem to play an important role. Furthermore, the initial antibody level and the maximum level of antibody reached after the streptococcal infection do not seem to be especially important.
3. The rheumatogenicity of streptococci prevalent at certain seasons or periods seems greater than

that of strains prevalent at other times. This variation in rheumatogenicity shows some correlation with the ability of the streptococcal organisms to cause an antibody response, but there also appear to be additional biological properties of the streptococcus which alter its rheumatogenicity. The nature of these properties is not known.

4. Finally, it would appear that over and above the patient's ability to produce an antibody response to streptococcal infection, there are additional host factors which determine an individual's susceptibility to rheumatic fever. The presence of such a host factor or factors is suggested by the fact that persons who already have rheumatic heart disease seem to develop recurrences of rheumatic fever following streptococcal infections more often than do rheumatic subjects who show no signs of heart involvement. The nature of the host factor which influences rheumatic susceptibility still remains unknown.

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# A New Type Of Glycogen Disease\*

S. VAN CREVELD

Since the first clinical and pathological description in 1928<sup>3,16</sup> and 1929,<sup>4</sup> glycogen disease or glycogen storage disease has often been the subject of intensive clinical and laboratory studies. Until 1932 only 2 types of glycogen disease were distinguished: the hepatomegalic or hepatonephromegalic type and the cardiomegalic or generalized type (Pompe). Better classification became possible when G. T. Cori<sup>1</sup> found in 1952 a specific enzyme defect, — absence of glucose-6-phosphatase, — in the hepato (nephro) — megalic type of disease. Since then, the enzymatic studies of cases of glycogen disease have been greatly extended, due to the work of the Cori's<sup>2</sup> and of Hers<sup>7</sup> especially.

Until a few years ago 4 types of glycogen disease could be distinguished. In recent years at least 2 more types have been added. Type V, in which there is an important increase of stored glycogen in the muscle and no reliably detectable phosphorylase in the skeletal musculature; and type VI, in which exists an elevated liver glycogen and marked deficiency of liver phosphorylase. The latter type was found by Hers when performing enzymatic studies of biopsy material of 3 young children with glycogen liver. He found a marked deficiency of liver phosphorylase and normal values for glucose-6-phosphatase (lacking or deficient in type I) and for amylo 1-6-glucosidase (lacking or deficient in type III). The glycogen content of the liver biopsy was very high in 2 children and normal in the third child. Lamy et al.<sup>9</sup> have made a further study of these three children. They all had an enlarged liver and the fasting blood sugar was low or normal; ketonuria was present in one patient. The epinephrine and glucagon test were slightly positive. After intravenous injection of galactose there was a normal increase of the glucose in the blood. The histologic study demonstrated a glycogen liver; the accumulated polysaccharid was normal.

Recently we observed a patient with glycogen disease in whom also Hers found a deficiency of liver phosphorylase in a liver biopsy. The patient was a boy nearly 2 years old; the fifth child of healthy parents. The other children are normal. A sixth child (the first born) died probably from a congenital heart. There was no consanguinity between the parents or the grandparents of the patient. No evidence of the presence of other cases of liver enlargement in the family. The mother took a normal diet during pregnancy and has been healthy during this period. Our patient was born spontaneously in normal time. Birth weight was 3.8

kg. Development during first year of life was normal; walking only was delayed. A very large liver was noted shortly after birth. When admitted to the pediatric clinic at the age of 22 months, his height was 81 cm, his weight was 12.3 kg. The fontanel was still open, slight rachitic symptoms were found. The skeleton was osteoporotic with delayed ossification. The heart and lungs were normal. Electrocardiogram normal. X-ray of heart normal. Liver palpable 7 fingers below the costal margin (Fig. 1). Spleen was not palpable.

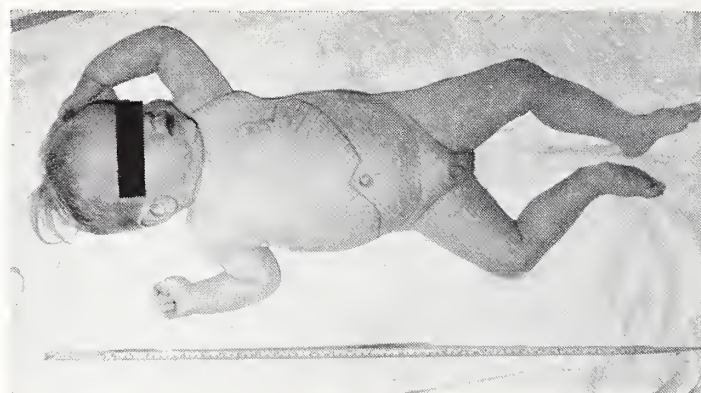


FIG. 1

Left testes was undescended. Urine: usually urobilin +, acetone usually slightly + to + in fasting state, negative during the day; reduction always negative.

Slight hypoglycemia in fasting condition  
(see below)

Glycogen blood increased: 19.7 mgr.%

Cholesterol serum 393 mgr.% (norm. 140-240)

Cholesterol ester 62% of total.

Lipid phosphorus 19.4 mgr% (norm. 7-14)

Triglycerides 446 mgr% (norm. 37-134)

Serum transaminases:

SGOT 72 U (norm. <40 U)

SGPT 33 U (norm. <35 U)

Total bilirubin: 0.2 mgr.%

Glucose tolerance test (15 gm orally):

→ Fasting bloodsugar 0.75 o/oo

→ After 1½ hours 1.08 o/oo

→ After 2½ hours 1.26 o/oo

Galactose-test: after 15 gm orally no excretion in urine

Thymol-turbidity 7 U.

Protein spectrum of serum:

→ total protein 7.2 gm/100 ml

→ albumin 3.96 gm/100 ml

→ euglob. 1.01 gm/100 ml

→ pseudoglob. 2.23 gm/100 ml

Clotting factors: slightly increased residual pro-

\*From the Pediatric Clinic of the University of Amsterdam, The Netherlands.

thrombin of serum and slightly prolonged prothrombin time; otherwise normal.

Biopsy of liver: glycogen considerably elevated: 11.9%, histologically typical aspect of glycogen liver. Enzymes (Hers): glucose-6-phosphatase normal: 2.1 (1.85 — 3.6),\* amylo 1.6-glucosidase: normal :0.15 (0.054-0.220).\*\* phosphorylase abnormally low! :3.3 (12-33).\*\*\*

Our patient is therefore an example of the type of glycogen liver reported for the first time by Hers in 1959 in 3 patients with a deficiency of liver phosphorylase.

Lamy et al. who made a clinical study of the 3 cases, investigated in their patients also the result of a number of tests, which normally are applied to recognize the different types of glycogen liver. Analogous investigations in a large number of patients have been done by Sidbury.<sup>15</sup>

Fasting bloodsugar in the 3 cases of Lamy were slightly decreased to normal. This corresponds with our findings. Ketonuria was constantly present in the first case of Lamy et al. In our case ketonuria was usually slightly + to negative. Abnormal cholesterol — and lipid-phosphorus values were present in the cases of Lamy et al. and also in our case. Uric acid of the blood was increased in one of the cases of Lamy et al. Of particular interest is the comparison of the results of other tests:

a. Epinephrine test:

In all 3 cases of Lamy et al administration of epinephrine had a negative result. In our case a positive result was obtained. Sidbury found impaired or a normal response in patients with type VI. As regards the results obtained by Lamy et al. we must remark that in their first and third case only the result of 2 determinations of bloodsugar during the first thirty minutes after injection of epinephrine are given and in their

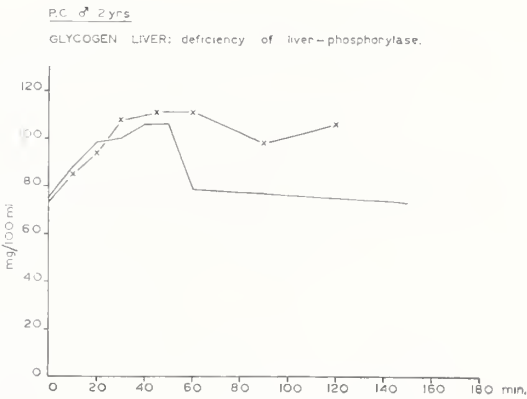


FIG. 2. Blood sugar after epinephrine ( $\frac{1}{4}$  ml. 1:1000 subc. —x—x—) and after glucagon (4/10 ml. i.v.—).

of Hers in the liver biopsy, is present, probably was sufficient to mobilize a certain amount of glycogen after epinephrine-injection.

b. The glucagon test has been performed only in 2 of the 3 patients of Lamy c. s. and in both cases the result was slightly positive. An analogous result was obtained in our patient (Fig. 2). (We gave 0.4 ml intravenously e.g. 0.7 ml pro M<sup>2</sup> bodily surface and compared the results with those obtained by Rossi<sup>12</sup> in healthy children.)

FASTING BLOODSUGAR		0.76 mg/ml
10' after glucagon injection		0.88 mg/ml
20' after glucagon injection		0.98 mg/ml
30' after glucagon injection		1.00 mg/ml
40' after glucagon injection		1.06 mg/ml
50' after glucagon injection		1.06 mg/ml
60' after glucagon injection		0.79 mg/ml
90' after glucagon injection		0.77 mg/ml
150' after glucagon injection		0.73 mg/ml

c. The intravenous galactose test of Schwartz et al<sup>13</sup> showed in all 3 cases of Lamy c. s. a considerable

FASTING BLOODSUGAR		0.73 mg/ml
10' after subcutaneous injection of 0.25 mgr. of epinephrine:		0.85 mg/ml
20' after subcutaneous injection of 0.25 mgr. of epinephrine:		0.94 mg/ml
30' after subcutaneous injection of 0.25 mgr. of epinephrine:		1.08 mg/ml
45' after subcutaneous injection of 0.25 mgr. of epinephrine:		1.11 mg/ml
60' after subcutaneous injection of 0.25 mgr. of epinephrine:		1.11 mg/ml
90' after subcutaneous injection of 0.25 mgr. of epinephrine:		0.98 mg/ml
120' after subcutaneous injection of 0.25 mgr. of epinephrine:		1.06 mg/ml

third case only 1 value is given during this period. Therefore it is not certain whether an *initial* elevation of bloodsugar after epinephrine-injection occurred. In our case there was a normal response to the epinephrine-injection (see also Fig 2):

We must conclude, that in our case the small amount of liver phosphorylase which, according to the finding

\*in brackets values obtained by Hers in 7 cases of type III and unknown.  
\*\*in brackets values obtained by Hers in 7 cases of type I.  
\*\*\*in brackets values obtained by Hers in 12 cases of type I and III.

increase of the glucose content of the blood. These were also the findings of Hers in our case (Fig. 3). This excluded a deficiency of glucose-6-phosphatase in the liver (type 1). We have performed some more tests in our patient, which have been recommended to distinguish glycogen liver from other illnesses with liver enlargement and which have also been considered for differentiation of the various types of glycogen liver.

d. The dihydroxyacetone test (DHA-test) has been recommended to distinguish glycogen liver from other diseases in which liver enlargement is involved.<sup>5,10,11</sup>

In type 1 there is no marked increase of the dihydroxyacetone content of the blood after administration of dihydroxyacetone orally, as in all other possible causes of liver enlargement.

The following results were found in two tests in our patient with phosphorylase deficiency in the liver:

Dihydroxyacetone 1 gm/kg/orally:

	Bloodsugar		Dihydroxyacetone	
	1st test	2nd test	1st test	2nd test
fasting	0.77 mg/ml	0.69	0.022	0.034
10' after administration	0.78	0.89	0.023	0.045
20' after administration	0.93	0.87	0.024	0.036
30' after administration	0.95	0.85	0.022	0.031
60' after administration	0.81	0.80	0.025	0.029
120' after administration	0.99	0.82	0.027	0.038
180' after administration	0.77	0.70	0.024	0.031

With respect to changes in the total bloodsugar as well as to the increase in the dihydroxyacetone content of the blood in this patient with phosphorylase deficiency in the liver, the results were the same as in case of glycogen liver type I, i.e. there was no significant increase in the level of dihydroxyacetone in the blood.

e. Recently Sidbury et al.<sup>14</sup> pointed out that the glycogen of the erythrocytes in type IV of glycogen liver should be low and abnormal and that in type I and II the glycogen content of the erythrocytes and structure of the glycogen should be normal. In type VI he found the red cell glycogen concentration to be elevated.

In our patient with phosphorylase deficiency we found a glycogen content of the erythrocytes, which 106 m gram per gr. hemogl. (norm: in children from 1 — 12 years old: 22 — 109 m gram).

f. Finally determination of the activity of the phosphorylase of the leucocytes has been determined in our patient and in some other patients, in whom the type of glycogen liver had been definitely established by Hers (Hulsmann, Oei and Creveld).<sup>8</sup>

It appeared that the activity of the phosphorylase of the leucocytes in our patient and in 2 other patients with glycogen liver due to phosphorylase deficiency in the liver, was markedly decreased. This was also the case in 1 of our patients with glycogen liver, in whom no enzyme determinations in a liver biopsy were performed.

It was remarkable that the activity of the phosphorylase of the leucocytes was also decreased in the mother of both our patients and in a sister of the second patient.

Two patients with glycogen liver due to deficiency of glucose-6-phosphatase in the liver had a normal activity of the phosphorylase of the leucocytes.

COMMENTS

The patient described is a new example of the hepatomegalic type of glycogen disease, due to defi-

ciency of liver phosphorylase, which was recently discovered by Hers. Clinically the patient has many symptoms in common with the patients with a glycogen liver due to a deficiency of other enzymes. The differences became more manifest in the results of the laboratory studies. In our case the fasting blood sugar is only moderately reduced, the tendency to

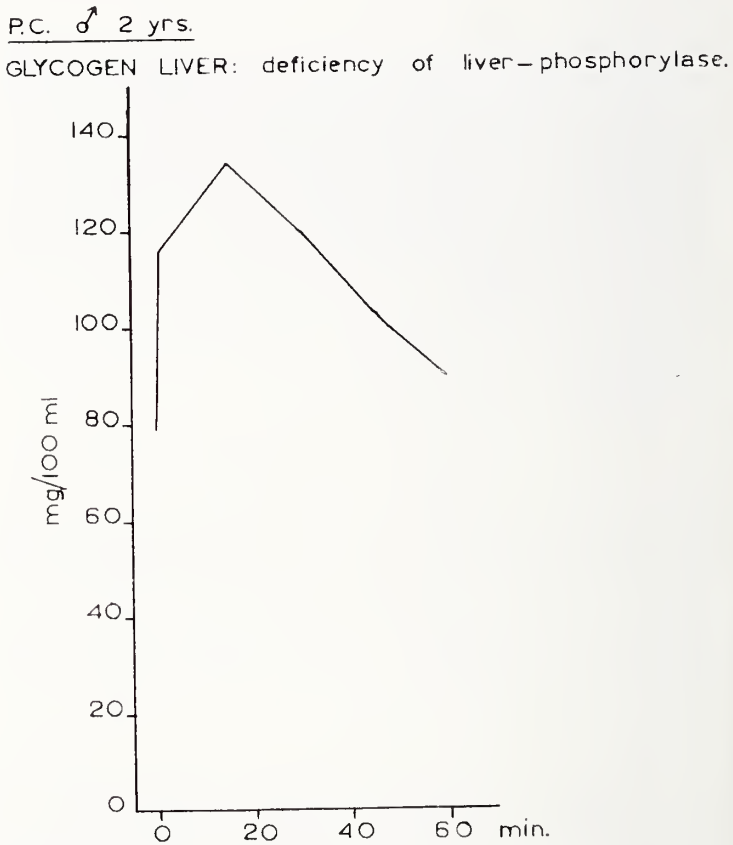


FIG. 3. Blood glucose after intravenous galactose (1/2 glkg).

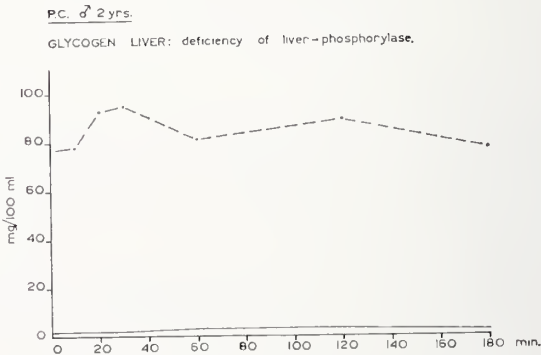


FIG. 4. Blood sugar (—.—.—) after lg. DHA/kg by mouth.

ketosis is smaller than usual in cases of type I of glycogen liver. The normal reaction to adrenalin as

found in our case, is practically never found in cases of type I or III. The weak reaction to glucagon more or less corresponds to the finding of Henion and Sutherland<sup>6</sup> according to which the phosphorylase of liver responds to glucagon.

The finding by Hulsmann, Oei and Van Creveld<sup>8</sup> of a deficiency of phosphorylase in the leukocytes of the patient and those of his mother and an analogous finding in another patient — in whom however no enzyme studies in liver biopsy have been made — deserves finally special attention. It may help to make a biopsy of the liver in such cases superfluous and further more it could perhaps increase our insight into the possible inheritance of this disorder.

#### SUMMARY

Detailed clinical and biochemical findings are communicated in a 2 year old boy with a glycogen liver, due to deficiency of liver-phosphorylase, a type of glycogen liver recognized for the first time by Hers.

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### ETIOLOGY OF THE STURGE-WEBER SYNDROME WITH CHROMOSOME ANALYSIS OF A CASE — *Continued from Page 79*



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### The Unclassified Mycobacteria

CHARLES H. OKEY, PH.D.\*

The necessity for cultural and biochemical studies of acid fast bacilli isolated from sputum and other body fluids has been emphasized in the last few years by the recognition of bacilli with acid fast staining properties which while not *M. tuberculosis* are nevertheless capable of producing human illness, in many instances indistinguishable from that of classical tuberculosis. These organisms have been termed by various authorities as "atypical," "anonymous" or "unclassified" Mycobacteria. It would seem appropriate to discuss the nature and significance of these organisms against the background of the procedures used in the Diagnostic Laboratory for the isolation, study and reporting of organisms isolated from specimens suspected of containing tubercle bacilli.

Sputum specimens are treated with sodium hydroxide which digests tissue and mucus and frees organisms trapped in these components. Also, most contaminating organisms are eliminated. Centrifugation of the digested material concentrates the remaining organisms. The supernatant is discarded and the sediment neutralized with acid. A portion of the sediment is spread on a slide for acid fast staining and other portions are planted on four tubes of modified Lowenstein-Jensen medium. The tubes are incubated at 36°C. for six weeks and examined at weekly intervals for growth. Smears stained by the Ziehl-Neelson method are examined for acid fast bacilli and for fungi. If the latter are found, sediment is planted on mycology media.

A report is forwarded to the physician at this point indicating the appropriate findings of the microscopic examination. Positive reports of "fungi found" are followed in six weeks by final reports indicating the results of mycological examination. Reports of "acid fast bacilli found" are based solely on microscopic observation and should not be equated with a report of *M. tuberculosis*. Isolating an acid fast bacillus in pure culture and studying its morphological and biochemical properties are the only accurate means of identifying the tubercle bacillus among the many acid fast bacilli. The National Tuberculosis Association summarizes this in the following statement published in Diagnostic Stand-

ards and Classification of Tuberculosis: "The demonstration of tubercle bacilli in clinical specimens is the one essential criterion in the definite diagnosis of active tuberculosis."

Colonies of suspicious morphology which develop on the Lowenstein-Jensen media tubes are stained by the Ziehl-Neelson method and those which are acid fast are examined for the following factors: cord formation (a measure of virulence), niacin production, catalase production and reaction to light in terms of pigment production. Reports on these findings indicate either negative results or a designation of the particular acid fast bacillus found. These various organisms are discussed in succeeding paragraphs.

*Mycobacterium tuberculosis, variety hominis*, is the classical "human type" and is responsible for 98% of the mycobacterial disease in the U. S. today. The organism has a characteristic rough colonial morphology, produces little if any pigment, forms cords and is both niacin and catalase positive except for those strains resistant to isoniazid which are catalase negative. In addition, the INH resistant strains are either not infectious for the guinea pig, or are of reduced virulence, which is in sharp contrast to the usual virulence of the human strain for this species. The bovine variety of *M. tuberculosis* is a rare cause of human disease in this country at the present time. Human disease caused by *M. avium*, the avian type of tubercle bacillus, has been documented in only a small number of cases since the organism was first described many years ago.

The "anonymous" or "unclassified" Mycobacteria are composed of a heterogeneous group whose etiologic role in human disease is being clarified at the present time. There are several unique characteristics that serve to set these organisms apart from the typical human tubercle bacillus: for example most, but not all, colonies are smooth and pigmented, show resistance to p-aminosalicylic acid in many strains and moderate resistance to isoniazid and streptomycin, and have reduced or lack virulence for the guinea pig and rabbit. None of the group produces niacin.

Several hundred cultures of these organisms from 50

\*Director, Diagnostic Laboratory

Veterans Administration hospitals and other sources were assembled and studied by Dr. E. H. Runyon of the Veterans Administration Hospital, Salt Lake City. His publication in 1959 established a grouping system based on pigmentation and speed of growth. This system is used throughout the country and is the basis of reporting by the Diagnostic Laboratory.

Group I — "Photochromogens": These grow slowly producing large smooth colonies which are non-pigmented when grown in the dark but become a deep yellow after brief exposure to light and reincubation overnight. All of these strains are pathogenic for mice. *M. kansasii* is the tentative name for members of this group.

Group II — "Scotochromogens": Members of this group grow slowly producing yellow to orange pigmented colonies in both dark and light incubation conditions. They have been implicated in lymph gland infections, especially in children, but only rarely have they been suspected as the cause of pulmonary disease in man.

Group III — "Non photochromogens": (Battey strains) These strains are mostly non-pigmented on primary isolation but occasionally tan, pink, or yellow colonies appear and still others develop pigment on subculture or after prolonged incubation. Many of the Battey strains resemble avian tubercle bacilli but tests are available to distinguish the two.

Group IV — "Rapid Growers": Most of these organisms grow to maturity in two to four days even on simple nutrient media. Many members of the group are saprophytes, some of which are already named. *M. fortuitum* is such an organism and has been firmly implicated in human disease.

Cultures having the respective characteristics of each of the first three groups have been isolated and reported by the Diagnostic Laboratory during the past six months. Some of the cultures have been from patients with chronic lung disease who have never had an isolation of *M. tuberculosis*. The presumption of the etiologic role of the particular unclassified mycobacteria is quite strong. The prevalence of disease or infection caused by these organisms in this state is not known. The National Tuberculosis Association estimates that from 0.5 to 2.0% of patients who are thought to have pulmonary "tuberculosis" are found to harbor various unclassified mycobacteria rather than true *M. tuberculosis*. Several states have accumulated much valuable information on the group particularly in the southeastern states where Group III infections appear to be widespread; Group I appears to be more frequent in other parts of the country.

A cooperative study between the Florida State Board of Health and the Florida Tuberculosis Hospitals directed at accumulating bacteriological, epidemiological and clinical data was begun in 1956.<sup>1</sup> Initially, cultures were taken only on persons known or suspected of having tuberculosis or on contacts of known cases but as

the study progressed and evidence of a wider spectrum of disease and even inapparent infection was found, the study was expanded to include a broader variety of clinical syndromes and persons without apparent illness. While the study is still in progress, certain preliminary findings are available. During the study, 1500 persons yielded unclassified mycobacteria and of this number two-thirds belonged to Group III and only 3% to Group I. Of the cases with available clinical data, one-third had an active pathologic process associated with multiple isolations of unclassified mycobacteria and another third had a questionable association with the unclassified group. The remaining third showed no evidence of disease association with the group. About 40% of the total cases yielding unclassified organisms had had a previous diagnosis of tuberculosis about two-thirds of which were culturally proven cases. Disease due to the unclassified group was observed predominantly in older age individuals and in males rather than females. Occupational groups working in close contact with the soil showed a higher incidence than other groups. The negro race was more frequently involved than the white race. New cases occurred more frequently in the spring than in other seasons of the year.

Exposure to Battey strains is apparently widespread in Florida. Skin sensitivity tests using a tuberculin type material prepared from the Battey strain showed a sharply rising rate of reactivity to the material in residents: 20% of twelve year old children were positive yet the rate reached a maximum of only 35% in the 50 year old group. Almost 4% of normal healthy children and young adults yielded Group III organisms from their sputum.

Efforts to determine the reservoirs of the organisms revealed that the bacilli are commonly found in soil, water and dust. Studies among household contacts of cases harboring anonymous mycobacteria indicated that person to person transmission does not appear to occur or if it does it is of a very low order of magnitude.

Considering generally the cases of anonymous mycobacterioses in the country, it is apparent that infection and disease are more widespread than realized only a few years ago. Pretreatment resistance of the bacilli poses a difficult therapeutic problem; surgical extirpation has been used to remove involved lung tissue. In most jurisdictions, cases of unclassified mycobacterial disease have been carried on the tuberculosis registry with the etiology specified. Cases have been kept isolated in tuberculosis sanatoria away from sources of infection by typical tubercle bacilli.

As more specimens are examined and unclassified bacilli recovered it will be possible to place the importance of the organisms in their true perspective for the State of Maine.

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# Necrologies

## LOREN F. CARTER, M.D.

1892-1962

Loren F. Carter, M.D. of Waterville, Maine died on January 25, 1962. He was Superintendent of the Northern Maine Sanatorium at Presque Isle for 40 years and had been a resident of Waterville for the past six months.

Dr. Carter was born in Bradley, Maine on January 3, 1892, the son of Frank L. and Phoebe Spencer Carter. He attended Colby College and graduated from Bowdoin Medical School in 1917.

Dr. Carter was a member of the Maine Medical Association, Aroostook County Medical Association, American Medical Association, American College of Chest Physicians and the American Thoracic Society. He was also a member of the Congregational Church of Presque Isle, an honorary member of the Rotary Club of Presque Isle, a veteran of World War I and a Mason.

Surviving are his widow, Mrs. Frances Carter of Waterville; three nephews, Stanley Carter of Bangor, Andrew Welch of Fryeburg, Roland Welch of Bradley; three nieces, Mrs. Helen Brown of Bradley, Mrs. Hortense Taft of Duxbury, Massachusetts and Miss Viola Carter of Holyoke, Massachusetts.

## JOSEPH A. DONOVAN, M.D.

1882-1961

Joseph A. Donovan, M.D. of Belmont, Massachusetts died on January 29, 1961.

Dr. Donovan was born in Houlton, Maine on November 14, 1882, the son of William and Ann Smith Donovan. He attended Bates College and was a graduate of Harvard Medical School in 1912.

He was a member of the Maine Medical Association and the Aroostook County Medical Association.

## EVERETT C. HIGGINS, M.D.

1880-1961

Everett C. Higgins, M.D. of Lewiston, Maine died on October 6, 1961. Dr. Higgins was born in Pittsfield, Maine, February 12, 1880, the son of the late G. W. and Leona Richardson Higgins. He attended the local grammar school and was graduated from the Maine Central Institute in 1899. Following this he entered Bates College in Lewiston receiving his degree in 1903. It is fitting to note that his Alma Mater awarded him an honorary degree of Master of Arts in 1950 in recognition of his outstanding work as a physician and for his unselfish devotion to his college, his hospital, and his community.

After leaving Bates College in 1903, he was principal and superintendent of the Bowdoinham, Maine, High School from 1903 to 1905 and principal of Guilford, Maine, High School from 1908 to 1910. During these intervening years, he also studied medicine at the Bowdoin Medical School and received his M.D. degree from there in 1911. Part of his medical student time during 1910 was spent at the Lying-in Hospital, New York, and Boston City Hospital Outpatient Department, Boston, Massachusetts. He was an intern at the Central Maine General Hospital from 1911 to 1912.

Dr. Higgins entered general practice in Phillips, Maine in

1912 and continued there for ten years. He belonged to the Mount Blue Lodge of Masons of Phillips. In 1960 he was honored as a "Country Doctor" by residents of the town of Phillips during that community's "Old Home Week."

Dr. Higgins married a talented Bates classmate, the former Hazel Donham of Hebron in 1915. A son, Harold, was born in 1918 at Phillips. His wife, his son, Lt. Col. Harold Higgins, his three grandchildren, two sisters, and two brothers survive Dr. Higgins.

Dr. Higgins moved to Lewiston in 1922 and opened his office for the practice of medicine in Lewiston at 149 College Street. He had been continuously in the practice of medicine in the Lewiston-Auburn community for thirty-nine years and had seen patients there until just a few weeks before his death.

Dr. Higgins worked diligently to improve his medical knowledge which he did by doing post graduate work at the Massachusetts General Hospital in 1930 and by spending a month each year from 1933 to 1939 taking a course at the Joseph H. Pratt Hospital in Boston. He had also attended the monthly post graduate teaching clinics at the Central Maine General Hospital since 1932.

Dr. Higgins held many responsible positions during his active medical years and was a past President of both the Franklin and Androscoggin County Medical Societies.

Dr. Higgins belonged to many professional and social organizations including the Maine Medical Association, the Androscoggin County Medical Association, Stanton Bird Club, Sons of the American Revolution, and was a member of the Board of Directors of the Sarah C. Frye Home for Aged Women. He was an enthusiastic member of the local Y.M.C.A. and for many years swam regularly each week in its swimming pool. The Androscoggin Historical Society was another of the organizations in which he was much interested. He has offered this latter society his often-used and well-remembered combination office chair and examining table, in event the society wished to accept it.

Dr. Higgins was active in local community affairs. Perhaps his greatest accomplishment in this field was achieved when he acted as General Chairman of the successful drive to raise funds to build the Stewart wing at the Central Maine General Hospital in 1947.

The October 1950 issue of the *Journal of the Maine Medical Association* was dedicated to Dr. Higgins and included a poem dedicated to him on his 70th birthday by the late Dr. Julius Gottlieb. This was one of the many tributes paid to him during his lifetime.

CHARLES W. STEELE, M.D.  
EUSTACHE N. GIGUERE, M.D.  
RALPH A. GOODWIN, SR., M.D.

## HENRY C. KNOWLTON, M.D.

1897-1962

Henry C. Knowlton, M.D. of Bangor, Maine died on January 7, 1962, after a long illness.

Dr. Knowlton was born in Knowlton, Quebec on November 13, 1897, the son of Paul Holland and Margaret S. Knowlton. He attended Guilford High School and received his medical degree from McGill University Medical School in 1923. Dr. Knowlton began practicing medicine in Hampden, Maine in 1923 and moved to Bangor in 1930.

He was a past President of the Penobscot County Medical Association, a member of the Maine Medical Association, American Medical Association and President of the Staff at the Eastern Maine General Hospital in the early 1940's where

he established the Blood Bank. Dr. Knowlton was active in Bangor municipal affairs serving two terms on the City Council during the war years and was twice elected Chairman of the Council.



DR. KNOWLTON

He is survived by two daughters, Mrs. Emerson P. Barrett, Lincoln, Massachusetts and Mrs. Kenneth DeCellis, Dallas, Texas; a son, Henry C. Knowlton, Jr., Hampden; a sister, Miss Sarah D. F. Knowlton, Bangor; two brothers, Paul Holland Knowlton, Schenectady, New York and Thomas A. Knowlton, New York City; five grandchildren and several nieces and nephews.

## ROLAND B. MOORE, M.D.

1886-1961

Roland B. Moore, M.D., well known Portland Obstetrician, died in Bethesda, Maryland on November 10, 1961. He was born in Portland, Maine, May 24, 1886, the son of William S. and Sarah Toner Moore. He was a graduate of Portland schools and of Bowdoin Medical School in the Class of 1907. He was Clinical Instructor at his Alma Mater from 1911 to 1920. In the last year of the Medical School (1920-21) he was Professor of Pediatrics.

After starting his practice in Portland he was appointed Adjunct Physician at the Maine General Hospital in 1919, and in 1920 Adjunct Obstetrician, and in 1925 Obstetrician. Prior to this there was no real Obstetrical Service, but Dr. Moore and Dr. Everett organized the Service in 1925 and in 1929 Dr. Moore was appointed Chief of Service, and Dr. Everett Assistant Chief. Dr. Moore continued in this capacity until 1946, when he was appointed to the Honor Staff and a year later to the Consulting Staff.

Dr. Moore watched over his Service with an exacting eye, and in his usual meticulous way. The Service grew and expanded from a few beds on old Ward C with a delivery room adjacent to occupying half of Ward E with the Gynecological Service on the other side. The duty period at that time was four months on Service followed by four months off. During this four months, he made rounds every day. Every patient was examined even though apparently doing well. Undoubtedly this particular care was responsible for practically no cross infection in the Ward.

Dr. Moore served in both World Wars, serving two years in France as a Major in World War I. When World War II was rearing its head, he organized the 67th General Hospital

from 1940 to 1941 from doctors and nurses of the Maine General Hospital with some others from the Central Maine General Hospital. As Colonel now, he served as C.O. of the 67th General Hospital until its demobilization in 1945. He was one of the few Reserve Officers who kept command during this war, as many of the other medical men who had organized units were replaced by Regular Army Officers before many months had passed.

On his retirement as Chief of Service in 1946, the Administrator of the hospital, Dr. Stephen Brown, wrote to Dr. Moore as follows: "The Obstetrical Service is truthfully one of your own creations because it has been through vision and untiring effort that you have brought it to a high point of efficiency. You also showed the same foresight and tenacity in organizing the 67th General Hospital. When you brought it to the Staff at first you did not get the full cooperation of the men and I did not think personally it could be done. This organization was an outstanding accomplishment in the hospital's long history — one to which we may always point with pride, thanks to your able leadership."

Dr. Moore was a member of the Portland Medical Club, Cumberland County Medical Society, Maine Medical Association and the American Medical Association. While living in Westbrook, he served two terms on the School Board. He was also President of the Prides Corner Kiwanis Club, and a life member of Ancient Landmark AF & AM. In 1956 he became Chief Emeritus of the Obstetrical Service and in 1960 went to Togus as Certification Officer.

PHILIP H. MCCRUM, M.D.

## C. EARLE RICHARDSON, M.D.

1885-1961

C. Earle Richardson, M.D. of Brunswick, Maine died in Portland on December 17, 1961, as the result of an automobile accident. He was 76 years old and had only recently retired from the practice of medicine, which he pursued vigorously for 50 years, the last ten of which saw him handicapped by chronic lymphocytic leukemia.

Born in Avon, Maine, on March 14, 1885, the son of David and Clara Frances Richardson, he was graduated from Bowdoin College in 1909. After a brief career in teaching he entered the Bowdoin Medical School, graduating in 1916. Following internship at the Maine General Hospital, Dr. Richardson became director of the Somerset Hospital in Skowhegan and practiced there until 1926, when he established in Brunswick a small hospital which for several years was the town's only hospital, complete with a school of nursing.

Aside from the pleasure of a long and honorable practice, Dr. Richardson enjoyed many offices and honors. He served as an officer of the Cumberland County Medical Association for several years and was once President of the Brunswick Rotary Club. In 1941 he became a member of the International College of Surgeons. He was a member of United Lodge AF & AM, St. Paul's Chapter, Mount Vernon Council, and DeMolay Commander of Skowhegan, Scottish Rite and Kora Temple Shrine and a charter member of the Skowhegan Rotary Club.

Ethel Pike Richardson, Dr. Richardson's wife for 52 years, died in 1960. Their only child died in 1945. Dr. Richardson was an active and devoted member of St. Paul's Episcopal Church in Brunswick and was the donor of the rectory of this church.

A brother, George of Jay, Maine, two aunts, and a niece survive Dr. Richardson.

His many friends and patients will recall C. Earle Richardson, M.D. as a generous man of courage and wisdom, with a fine feeling for the humorous side of things.

STANLEY E. HERRICK, JR., M.D.

# County Society Notes

## KNOX

Twenty members and two guests were present at the Knox County Medical Association meeting which was held at the Knights of Columbus Hall in Rockland, Maine on February 13, 1962.

The business meeting followed the social hour and dinner. William A. McLellan, M.D., President, conducted a discussion of Blue Shield for the aged.

Harold L. Osher, M.D. of the Maine Medical Center presented a very interesting talk on cardiac catheterization.

MUSTAFA V. ONAT, M.D.  
*Secretary*

## HANCOCK

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on March 14, 1962.

Robert F. Russell, M.D. reported relative to a recent meeting of the Maine Medical Association Health Insurance Committee which was followed by a spirited discussion on the pro's and con's of the proposed Blue Shield Plan for those over 65 and Blue Cross-Blue Shield coverage in general.

Edward J. Hughes, Jr., M.D. of Bangor presented an interesting case report of a recent peritoneal dialysis he had done for salicylate poisoning and a prepared talk on "The Use of Antibiotics in the Newborn."

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## YORK

Twenty-six members and four guests were present at the York County Medical Society meeting which was held at the Webber Hospital in Biddeford, Maine on March 14, 1962.

The meeting was called to order by the President, Marcel D. Ouellette, M.D., following a social hour and dinner. Germain R. Binette, M.D. of Biddeford was elected to membership in the society.

Cornelius E. Sedgwick, M.D. of the Lahey Clinic gave a very interesting talk on "Portal Hypertension" with lantern slides.

CHARLES W. KINGHORN, M.D.  
*Secretary*

## CUMBERLAND

A meeting of the Cumberland County Medical Society was held on March 15, 1962 at the Eastland Motor Hotel in Portland, Maine. Ninety members and guests were present.

After a social hour and dinner, the meeting was called to order by the President, Robinson L. Bidwell, M.D. Stanley G. Dienst, M.D. and Clement A. Hiebert, M.D. of Portland and Kathleen M. A. Millard, M.D. of Windham were elected to membership in the society.

The secretary gave a report of the Health Insurance Committee meeting which was concerned with the new Blue Shield Plan for senior citizens, as well as the new group, BSC contract for the members of the Maine Medical Association.

S. Allan Howes, Chairman of the Citizens Survey of the

United Community Service of the Portland area, was the guest speaker. Mr. Howes gave a description of the organization and work of the survey in respect to its health aspects. This was further discussed by Donald F. Marshall, M.D., who was the medical consultant for the survey, as well as Edward G. Asherman, M.D., who represented the United Community Services. It was recommended that the county society Public Health Committee study the health recommendations and report its opinion to the county society for action. There was further discussion by David Davidson, M.D., District Health Officer for Cumberland and York, Philip P. Thompson, Jr., M.D., Boris Vanadzin, M.D., Health Officer for Portland, Mr. Barker of the Maine Medical Center, among others. Thomas A. Martin, M.D., Councilor for York and Cumberland counties, made some pertinent remarks about the difficulties of implementing some of the recommendations, as well as the fragmentation of medical care caused by the formation of new organizations for each disease. He further spoke about the AMA's education foundation and recommended that the members of the county society give first priority to the Maine Medical Association's fund which so far totals \$32,000.

ALBERT ARANSON, M.D.  
*Secretary*

## PENOBSCOT

A meeting of the Penobscot County Medical Society was held at the Tarratine Club in Bangor, Maine on March 20, 1962. There were fifty-five members present with the President, Clement S. Dwyer, M.D. presiding. James A. MacDougall, M.D., President of the Maine Medical Association, was a guest.

Raymond M. P. Donaghy, M.D., Professor of Neuro-Surgery from the University of Vermont Medical School, gave a most interesting lecture on "Cerebral-Vascular Disease."

At the business meeting, Deane L. Hutchins, M.D., University of Maine, Orono, Maine was welcomed as a transfer member from the Lincoln-Sagadahoc Society. Leonard G. Miragliuolo, M.D. pointed out the benefits of the proposed Blue Cross-Blue Shield coverage for physicians. He also presented material concerning the program for medical care of the aged.

Dr. Dwyer stated that a questionnaire concerning Social Security had been mailed to 92 members and that replies have been received from 89. The questions and answers follow:

1. Are you now paying for or receiving benefits under Social Security?  
15 "yes", 72 "no"
2. As a physician, do you want to be covered by Social Security?  
A) Voluntary — 64 "yes", 16 "no"  
B) Compulsory — 19 "yes", 18 "no"
3. If a voluntary plan is unacceptable to the United States Government, would you accept a compulsory Social Security plan?  
66 "yes", 20 "no"

From the above figures it will be noted that parts of some of the questions were not answered. The most important thing concerning the survey is that 89 out of 92 completed the form and that in reply to the last question, which is the most important, three and one-half times as many people would accept Social Security as would refuse if they had a chance.

FREDERICK C. EMERY, M.D.  
*Secretary*

# LOMOTIL®

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## ANTIDIARRHEAL TABLETS and LIQUID

**lowers motility / relieves cramping / controls diarrhea**

Roentgenographic studies by Demeulenaere<sup>1</sup> established that a single dose of 10 mg. of Lomotil slowed gastrointestinal transit within two hours and that it maintained its decelerating activity for more than six hours.

In diarrhea this lowered propulsion permits a physiologic absorption of excess fluid, lessens frequency and fluidity of stools and gives safe, selective, symptomatic control of most diarrheas. Concurrently, it conserves electrolytes and controls cramping.

Investigators have found the antidiarrheal action of Lomotil not only "excellent"<sup>2</sup> but "efficacious"<sup>3</sup> where other drugs have failed. . . ."

**DOSAGE:** For *adults* the recommended initial dosage is two tablets (2.5 mg. each) three or four times daily, reduced to meet the requirements of each patient as soon as the diarrhea is under control. Maintenance dosage may be as low as two tablets daily. For *children* daily dosages, in divided doses, range from 3 mg. (½ teaspoonful three times daily) for infants 3 to 6 months to 10 mg. (1 teaspoonful



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**NOTE:** Lomotil is an exempt narcotic preparation. Descriptive literature and directions for use detailed in Physicians' Product Brochure No. 81 available from G. D. Searle & Co., P. O. Box 5110, Chicago 80, Illinois.

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# Announcements

## Medical Films

The following films will be shown at the Pineland Hospital and Training Center on Thursdays at 11:00 a.m. in the Conference Room of the Treatment Building:

April 19, 1962 — "Medical Genetics - Part I," 34 min. color and "Medical Genetics - Part II," 30 min. color. The films were prepared by Victor McKusick, M.D. and his associates in the Division of Medical Genetics, The Johns Hopkins University School of Medicine.

April 26, 1962 — "Dwarfism," 40 min., Dr. S. Z. Levine, Professor of Pediatrics, Cornell University Medical School, and Pediatrician-in-Chief, the New York Hospital, presents a film clinic on dwarfism. "Modern Concepts of Epilepsy," 24 min., color. Discusses the nature, diagnosis, and treatment of epileptic disorders, using clinical cases to illustrate the various forms of the disease.

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Applications for certification in the American Board of Obstetrics and Gynecology, new and reopened, for the 1963 Part I Examinations are now being accepted. Candidates are urged to make application at the earliest possible date. The deadline date for receipt of applications is *July the first, 1962*.

All applicants and candidates for re-examination are required to submit with their application or letter of request, a *duplicate* list of their hospital dismissals for the preceding twelve months, made up in accordance with the revised format shown on the last page of the current Bulletin.

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No. 5

## Abdominal Wound Disruption Its Incidence, Prevention And Treatment

EUGENE E. O'DONNELL, M.D., F.A.C.S.\*

Mason has stated, "The urge to heal is almost as great as the will to live." Anglem defines wound disruption as "a separation of the peritoneal, muscular, and fascial layers of the wound," and uses the terms disruption and dehiscence synonymously. Moore distinguishes between these terms. He points out that wounds usually heal to completeness quite readily in the face of pre-operative starvation, negative nitrogen balance, and in the presence of infection. Brettauer has been credited with the first report on wound disruption in the American literature in 1889. Early estimates of the mortality from this condition varied between 20% and 80%. In 1909, Charles H. Mayo, at a meeting of the Western Surgical Society in Omaha, in discussing a presentation by Ries, brought out the fact that simple abdominal distention produced an internal pressure beneath the suture line, thereby causing anemia of the tissues about the wound, with resulting interference with wound healing. In a search of the literature of the library of the New York Academy of Medicine in 1911, Robert Morris was unable to find any reference to this subject. Crandon and Ehrenfried, in 1912, emphasized the importance of sepsis in preventing the adhesion of wound edges. Howes and Harvey, in a series of publications over a period of many years, a few of which are quoted here, studied the physiologic process of wound healing under various conditions, the tensile strength of suture materials, and of the holding tissues themselves, under normal con-

ditions and in the presence of infection. They demonstrated the unpredictability of the rate of wound healing in the presence of infection and other abnormal conditions. It was largely through their influence that the present custom of employing suture material, to quote the senior author, "suited to the needs of the patient rather than to the strength of the operator" became common practice. A continuation of these observations by Howes, et al, has resulted in approximately 70 publications on this and related subjects. Dunphy and Udupa have more recently made pertinent observations on the physiology of wound healing.

In 1950, Wolff studied 1700 consecutive abdominal operations. He found that wound disruption had occurred in 45 cases, a rate of 2.6%. The mortality rate in his series was 11.1%. He concluded that, in most instances, a combination of many factors was operative. He was unable to confirm the general impression that any technique of wound closure was entirely foolproof, or that any particular type of incision would prevent this complication. His report contained a critical analysis of the factors under indictment and recommendations for prophylaxis.

In 1961, Anglem reported a study of 1728 abdominal operations, with 6 wound disruptions and 1 death. There were 758 vertical upper abdominal incisions in which the multiple layer closure was reinforced by stainless steel wire through all layers, a technique which he recommends. In this group there were 3 wound disruptions, or 0.39%. Holman and Eckel have recommended a similar type of wound closure.

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The following factors have been considered by various authors as potential agents in this complication:

- Age
- Sex
- Race
- Presence of cancer
- Obesity
- Anemia
- Protein depletion
- Vitamin C deficiency
- Catgut sensitivity
- Type of incision
- Technique of wound closure
- Anatomical dissection of the abdominal wall
- Early ambulation
- Abdominal distention
- Respiratory complications
- Wound infection

In a study of surgical practice at the Mercy Hospital during the years 1952 to 1961 inclusive, 8249 major abdominal operations were performed. In order to conform to Anglem's series, the following procedures were excluded:

Inguinal hernia	1283
Femoral hernia	102
Umbilical hernia	105
Lumbar sympathectomy	58
Colostomy	104
Hiatus hernia	50
Abdominoperineal resection of rectum and sigmoid	82
<b>TOTAL</b>	<b>1784</b>

There were, therefore, 6465 cases for consideration at this time, including:

Gastric surgery	461
Biliary tract surgery	981
Colon resection	268
Miscellaneous	381
Splenectomy	25
Closure of colostomy	50
<b>TOTAL UPPER ABDOMINAL SURGERY</b>	<b>2166</b>

CORRECTED TOTAL OF ALL ABDOMINAL OPERATIONS,  
MERCY HOSPITAL:<sup>1</sup> 6465  
TOTAL WOUND DISRUPTIONS: 30  
PERCENTAGE OF WOUND DISRUPTIONS: 0.46%  
UPPER ABDOMINAL OPERATIONS: 2166  
NUMBER OF WOUND DISRUPTIONS IN UPPER AB-  
DOMINAL SURGERY: 20  
PERCENTAGE OF WOUND DISRUPTIONS IN UPPER  
ABDOMINAL SURGERY: 0.92%

<sup>1</sup>Eight-hundred and seventy four of the author's cases are included in this category with two wound disruptions, a perforated duodenal ulcer repaired through a vertical incision, and a total hysterectomy performed through a previous low midline incision.

It is apparent that in this study, the wound disruption rate in upper abdominal surgery was approximately twice as great as the incidence of all cases, and four times as great as the incidence in lower abdominal surgery. One patient in this series died. Although this study is too small to be of statistical significance, the mortality rate was 3.3%. Two patients disrupted their incisions on the 8th postoperative day at home, after the sutures had been removed prior to discharge from the hospital. One patient suffered wound disruption on the 11th postoperative day.

There is general agreement that a sanguinous discharge from the operative incision is pathopneumonic of this condition. Other symptoms and signs include pain, vomiting, sudden onset of distention, varying degrees of alteration in the patient's general condition, and gross evidence of disruption on inspection of the wound.

Abdominal incisions in our time are the product of several surgical specialties and surgical philosophies. We may sometimes be disposed to concern ourselves primarily with the technical details of alleviating the intra-abdominal disease, and ascribe faulty wound healing to bad luck rather than to technical imperfections in wound closure. While the peritoneal cavity will tolerate gross infection to a degree, the abdominal incision is more vulnerable to this complication. For example; on occasion, in the elderly debilitated individual where the incidence of wound disruption has been shown to be almost 1% in this study, we might be influenced to drain the acutely inflamed gall bladder through a minimum incision rather than perform a cholecystectomy through more adequate exposure. In hernial defects from previous operations in the case of the pendulous abdomen, apposition of tissues may be maintained in spite of tension, in some instances, by far and near mass sutures of steel wire, or some similar bacteria-repellent material, particularly if the patient has lost weight prior to the operation. We believe, when practical, this procedure is to be preferred to the introduction of tantalum mesh and similar substances in abdominal wall defects. The prophylactic use of antibiotics in clean wounds and uncomplicated cases must not be confused with their employment to attenuate the growth of bacteria in the presence of gross wound contamination, in surgical shock, or in failing circulation from any cause. The protection of skin edges, while sufficient in clean cases, should be supplemented by the protection of the entire raw surface of the wound, when practical, in the presence of intra-abdominal infection. Clean instruments and clean gloves for the closure of the abdominal incision in all potentially infected cases such as suppurative cholecystitis, ruptured appendix, gastrointestinal resection, and total hysterectomy, add further safeguards. In the stout individual with a wide costal margin, the transverse or modified transverse incision may be ideal for some types of elective upper abdominal

surgery. However, in a thin individual with a narrow costal arch, and perhaps in a person who has a low abdominal scar from a previous laparotomy, the vertical upper abdominal incision may be preferred. While the gradual evolution of surgical technique which has taken place during the past quarter century implies adequate hemostasis, approximation without tension, including a minimum amount of tissue in the suture line, and relatively fine suture material, it is, nevertheless, desirable that we do not allow this practice to become our master rather than our servant.

It is the author's distinct impression that there is a tendency to fail to use stay sutures when indicated in some instances, to place them improperly, or to remove them too soon, as illustrated from the cases reported here and from reports of this complication in the surgical literature. Removal of the sutures may easily become a ritual in the patient's convalescence, practiced both for the benefit of the patient and the surgeon. It marks a milestone in the postoperative course, and cements the professional relationship by implying that the operation has been a success. Removal of stay sutures is also sometimes practiced in order to remove a source of irritation in an incision which shows some inflammatory reaction. It is sometimes done without sufficient regard as to the reason the sutures were put there in the first place. The infected wound is a wound in which the healing process is delayed; therefore, this custom should be reversed, and will not materially delay the healing process, provided the stay sutures are bacteria-repellent.

In 1941, Glenn quoted Whipple, Meleny and Howes who, on or about that time, discouraged the use of stay sutures as promoting infection. It is my understanding that these authors no longer necessarily adhere to this concept. If the stay sutures are of such material that they are not invaded by bacteria, if they are placed in such a manner that hematomas do not form in the abdominal wall from the site of their insertion, and if, should this problem occur, the offending suture is immediately removed and repositioned, a great deal of the objection to their use is untenable. The author is in general agreement with Anglem in his recommendations as to the techniques of wound closure, but feels that the type of incision should be individualized.

Wolff has emphasized the necessity for cooperation between the anesthesiologist, in performing the toilet of the tracheo-bronchial tree, and the surgeon during the procedure of wound closure. I feel that it is likewise highly desirable that, with the naso-gastric tube in place, the stomach should be collapsed before the peritoneum is closed. In the early postoperative period, I believe that no amount of Compazine® or Compazine—like drugs, or Pituitrin or its derivatives should supplant naso-gastric suction, attention to the respiratory tract, and maintenance of proper fluid and electrolyte balance.

The secondary closure of the disrupted abdominal incision, no doubt, may call for some individualization, depending upon the extent of the problem and the condition of the patient. There may be many considerations which make the mode of handling this complication controversial. However, I believe that it is, in most instances, true that the sooner the abdominal viscera are returned to the peritoneal cavity and the wound edges placed in proper approximation to contain these viscera, the sooner normal convalescence will be resumed. Treatment of a minimum disruption, in the hope that one can settle for an incisional hernia which may be repaired later, may be questionable practice. In profound shock from a dilated stomach and/or intestine, or tension on the small bowel mesentery with consequent interference with its blood supply, the problem will not be solved by adhesive strapping or abdominal binders. However critical the patient's condition may be under these conditions, the risk of adequate anesthesia, carried out under proper conditions, may be less than that of expectant treatment and attempt at supportive measures which do not solve the basic problem.

	No. Operations	No. Wound Disruptions	% Wound Disruptions	Mortality
Wolff	452	21	4.64%	11%
Hull & Hankins	1292	39	3.01%	
Tweedie & Long	6588	62	0.94%	
Bettman & Kobec (1)	7500	32	0.43%	37.5%
Bettman & Kobec (2)	11694	60	0.51%	20%
Anglem & Gray	1728	6	0.39%	17%
Mercy Hospital	6465	30	0.46%	3.3%

SUMMARY

Closure of the abdominal incision presents a unique problem not present in many types of wound closures, in that tension on the suture line may be present to a greater degree than in almost any other type of incision, and that the failure of primary wound healing may be fraught with serious consequences.

Pertinent literature on the subject of wound disruption has been reviewed.

Statistical results of several institutions have been evaluated.

A report of the incidence of wound disruption at the Mercy Hospital over a ten year period is included.

These studies would indicate the present disruption rate to be somewhere in the neighborhood of 0.5%.

Factors which may contribute to this complication have been enumerated.

Comments have been made regarding secondary wound closure.

The treatment of wounds, in the light of increasing knowledge and experience, has been subjected to changing concepts, and as Harvey has said, "The treatment

*Continued on Page 111*

# Uncommon Causes Of Gastro-Intestinal Hemorrhage

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JOHN KNOWLES, M.D.,† PETER O'HARA, M.D.,† THOMAS CONEEN, M.D.†

The management of an undiscovered cause of alimentary hemorrhage even after a careful work-up remains a difficult problem that is familiar to most physicians.

This presentation includes a discussion of this problem, a brief report of 3 unusual cases of gastro-intestinal bleeding, and recommendations for handling these problems.

Read, Jones, and Stubbe<sup>1</sup> recently reported that in all large series of cases of alimentary bleeding studied, the cause of hemorrhage was obscure in 15-25% of the patients.

These authors followed 142 patients with undiscovered cause of bleeding for from three to seven years. One hundred and nine of the cases had no subsequent etiology for the hemorrhage and must remain a diagnostic mystery. The remaining 33 cases eventually showed the following lesions as causes for bleeding: peptic ulcer, cancer of the stomach, cancer of the small and large bowel, carcinoma of the pancreas, Meckel's diverticulum, telangiectasis of the intestinal mucosa.

Rare causes of gastro-intestinal bleeding that have been reported in the literature by different sources may be listed as follows: pseudoxanthoma elasticum, periarteritis nodosa, food allergy, Henoch-Schonlein disease, chronic nephritis, malignant hypertension, myelomatosis, amyloidosis, hemophilia, thrombocytopenia, von-Willebrand's disease. More specific lesions within the gastro-intestinal tract itself have been likewise reported as rare causes of alimentary hemorrhage such as: Mallory-Weiss syndrome, hemobilia, rupture of abdominal aortic aneurysms, leiomyoma, leiomyosarcoma, carcinoid, hemangioma, diverticulum of the large bowel.

The best way to manage patients with unexplained gastro-intestinal bleeding, after careful clinical laboratory and x-ray study, is to follow them for an undetermined period of time. Repeat examinations periodically seem to be in order, and the use of any other procedure not previously used should be employed to uncover the source of bleeding. Many of these cases will become candidates for exploratory operation, especially since any undiscovered lesion of the alimentary

tract causing hemorrhage may continue to bleed, and a high percentage of these lesions may be malignant. The incidence of malignancy has been reported to be as high as 20%. The value of exploratory laparotomy in these cases of obscure bleeding has been studied recently by a group at the Mayo Clinic.<sup>2</sup> Exploration was performed in 100 consecutive cases. A negative result was obtained in 53 of the procedures. Forty-eight of these cases were followed for a period of time, and it was found that 27 cases continued to lose blood post-operatively.

If abdominal exploration is undertaken in these types of cases, it must be done with a knowledge of possible negative findings in 50% of the cases. It must be done with meticulous care, having a high index of suspicion for the rare and uncommon causes of bleeding already mentioned. One should expect to derive the best results from the middle or older age group of patients, and from those cases which bleed severely. Gastrotomy at the time of exploratory is a worthwhile procedure when indicated, but blind gastrectomy should be avoided as it may fail to control bleeding in 57% of the cases.

## CASE REPORTS

*Case No. 1.* A female, age 44. The patient was first seen at the age of 34 with a history of massive upper G.I. hemorrhage. At this time the x-ray studies of the upper gastrointestinal tract revealed a small solitary diverticulum of the duodenum. No other abnormality was encountered. During the next nine years this patient was admitted to the hospital on fourteen different occasions three of which were for episodes of massive upper G.I. bleeding. Although the patient was thoroughly and repeatedly investigated never was a cause for her G.I. bleeding found. She had several upper G.I. series, repeat barium enemas, at least two gallbladder series and an I.V.P. During this interval as well, the patient reported that she did have spells of diarrhea and black stools. The only other significant finding in the history of this patient is that she had been suffering with a considerable amount of back pain for a number of years and had been under the care of an orthopedic specialist for this backache. The condition was thought to be due to osteoarthritis of the mid dorsal vertebra. On her last admission it was decided that owing to the fact

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that the cause of bleeding was still not determined the patient should be offered an opportunity to have a laparotomy. This was carried out and after careful examination of the entire G.I. tract and the abdomen as well no cause was found for the bleeding. Post-operatively the patient made an uneventful recovery. Although it was known that the patient had been taking aspirin for her backache for a number of years it was not until after her negative exploratory that evidence was uncovered that she had been able to associate with G.I. bleeding the periodic ingestions of large amounts of aspirin. This was a significant finding. The patient was discharged from the hospital and was advised not to take aspirin in any form.

#### COMMENT ON CASE NO. 1

Although it was known before exploratory operation that this patient did on occasion ingest large doses of aspirin, it was not considered of importance. However, shortly after exploration the finger of suspicion strongly pointed towards acetylsalicylic acid, and the patient was strongly advised against the use of acetylsalicylic acid in any form. The patient has been followed for several years and there has been no further evidence of gastro-intestinal bleeding.

There is still controversy over the exact mechanism of gastro-intestinal bleeding associated with the ingestion of acetylsalicylic acid products. However, there is evidence at hand which indicates that the quantity of blood loss is related to the total dosage of the drug, and the frequency of its administration. Investigations by Pierson et al<sup>3</sup> revealed that the ingestion of aspirin tablets which are enteric coated did not significantly influence the rate of bleeding nor did hypoprothrombinemia, or concomitant ingestion of milk or food. Weiss et al<sup>4</sup> studying the effects of aspirin on the gastric mucosa, observed gastroscopically 30 patients and found that 43% of them reacted to the administration of aspirin with congestive and hemorrhagic changes of the gastric mucosa, especially around areas in contact with, and in close proximity to, the drug.

*Case No. 2.* A 72 year old widow was admitted to the Hospital with a chief complaint of progressive weakness and passage of black colored stools. The patient stated that she had been in apparent good health until three days prior to admission when she developed progressive weakness associated with passage of many black bowel movements, and on three occasions black vomitus. She had never noted black stools in the past and denied any prior gastro-intestinal symptomatology.

Physical findings were limited to anemia and mild hypotension. Hematocrit on admission was 24%. All other laboratory work including serum bilirubin were normal. Over the course of the next three days her hematocrit rose to 33% with the aid of four pints of blood. Upper G.I. series suggested a gastric ulcer in the lesser curvature of the stomach. On December 9,

1955, an abdominal exploration was performed. Subtotal gastrectomy was planned but on exploration the stomach and duodenum appeared normal but there were multiple adhesions in the region of the distended gallbladder and a huge stone was seen in the common duct. The stone was removed from the dilated duct. The cholecysto-choledochal cyst was corrected by removing the gallbladder and including the fistulous opening in the common duct with the closure of the choledochostomy around the T. tube.

#### COMMENT ON CASE NO. 2

This case was followed for about 4 years until the time that the patient died of an unrelated cause. She never demonstrated gastro-intestinal bleeding after the operation described. From what was found at the time of the operation it was apparent that the stone had eroded through the gallbladder into the choledochus and by pressure necrosis was causing ulceration and hemobilia.

The term hemobilia as described by Sandbloom<sup>5</sup> originally in 1948 has been broadened today so that most authors refer to two types now, namely, the non-traumatic and the traumatic.

The following is a list of some of the non-traumatic causes of hemobilia:

1. Tumors of the liver, gallbladder and bile ducts
2. Acute hemorrhagic cholecystitis
3. Infarction of the wall of the gallbladder
4. Ulceration of the gallbladder or biliary ducts by stones
5. Aneurysm of the hepatic artery
6. Cavernous transformation of the portal vein

*Case No. 3.* A white male, age 78, was admitted for congestive failure and found to have anemia, hemoglobin 6.6 gms., hematocrit 21%. History revealed tarry stools and no bright red blood. Physical examination negative except for an inguinal hernia, indirect and hemorrhoids. Review of systems was negative. Work-up included stools which showed 2+ occult blood in each of three specimens. Prothrombin time, bleeding time and clotting times normal. Barium enema and G.I. series were negative. After seven weeks he was discharged; the cause of anemia was undetermined.

Fifteen months later he was readmitted because of weakness. Hemoglobin 6.8 gms., hematocrit 23%, tarry stools, 3+ occult blood. At this time sigmoidoscopic examination was negative except for internal and external hemorrhoids. Barium enema negative. G.I. series and fluoroscopy of chest and esophagus were negative. The patient refused any treatment for hemorrhoids, hernia or laparotomy and was discharged. Ten days after discharge, he returned to the O.P.D. pale and weak, hemoglobin 7.1 gms., hematocrit 22%. A battery of liver function tests was normal. A gastric analysis was done. A fasting specimen showed no free HCl, total acid 8 degrees and a trace of occult blood. The

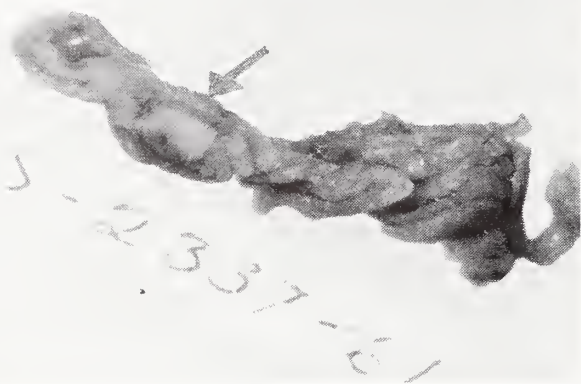


FIG. 1. Cut section through ileum. Arrow indicates white thickened area of muscularis.

patient was discharged after receiving transfusions, sufficient to restore blood loss.

He was home for only six days when he returned, his hemoglobin had fallen to 6.8 gms. and hematocrit 23%, from 9.7 gms. and 31% in 6 days. A G.I. Series was interpreted as question of varices in the fundus of the stomach, small direct sliding hiatus hernia. An esophagoscopy and gastroscopy revealed mild esophagitis and possible bleeding in the upper part of the hiatus hernia. In view of this a Levine tube was placed in the stomach; the contents aspirated showed 1 to 2+ guaiac, no evidence of gross blood on microscopic examination. An exploratory laparotomy was performed. The patient's liver was normal. The hiatus of the diaphragm would admit two fingers. Blood was visualized in the terminal ileum and hard button like nodules were palpable in the distal ileum. The involved segment was resected and a primary small bowel anastomosis was done.

**Gross Examination:** — The specimen received in the laboratory was a 41 cm. segment of ileum. Scattered along the mucosa were numerous very hard slightly elevated lesions with depressed central portions. The largest measured 1.5 cm. in greatest diameter. The lesions were more striking on palpation than by visual inspection. They felt like buttons in the muscularis. On section hard white or yellow tissue extended into the muscularis. Some extended close to the serosa — Fig. 1. Grossly these were suggestive of tumors arising in the muscularis possibly fibromas, leiomyomas or carcinoids.

Microscopically one sees clumps of well differentiated orderly cells with clumps of chromatin in the nuclei. The cytoplasm is poorly defined. The histologic pattern is typical of carcinoid. Figs. 2 and 3. The amount of invasion of the muscularis varied. In some the tumor cells invaded just beneath the mucosa. In others tumor traversed the muscularis to the serosa. In some sections small ulcerations of the mucosa with hemorrhage into the surrounding submucosa suggested that this man was indeed bleeding as a result of the carcinoids. Tumor cells were found in lymphatics leading to a diagno-

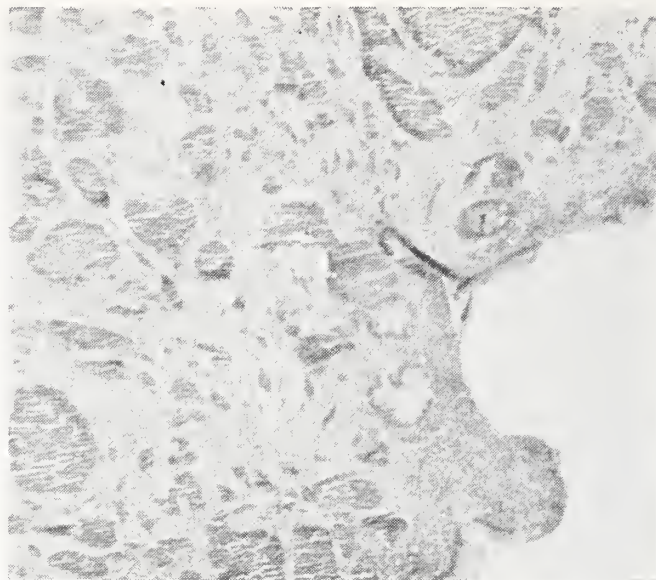


FIG. 2. Nests of carcinoid cells invading below mucosa. (75x)

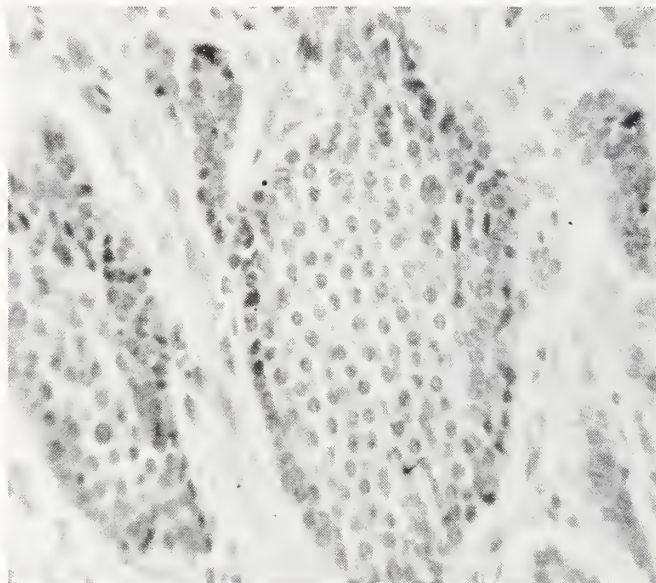


FIG. 3. Nests of orderly carcinoid cells. (x356)

sis of multiple malignant carcinoids of the ileum. Lymph node metastases were not present.

#### COMMENT ON CASE NO. 3

The work of Masson and colleagues showed for the first time that intracytoplasmic granules in the tumor cells had the ability to reduce silver salts. They stated that the tumor cell arose from a type of cell normally found in the gastro intestinal tract from the stomach to the rectum. It is known by many names, the most common being argentaffin or Kultschitzky cell.

Pearson and Fitzgerald analyzed cases of carcinoid seen at the Boston City Hospital from 1938-1948 and combined their series with one previously reported by Porter and Whelan covering the years 1910-1937.<sup>6</sup> One-hundred and forty cases of carcinoid were found. Of these 98 were of appendiceal origin, 42 of non-appendiceal origin. Of the non-appendiceal group the ileum was most commonly involved. All other regions of the gastrointestinal tract were involved to a lesser

degree. Metastases occurred in none of the appendiceal carcinoids. The non-appendiceal group showed metastases in 38% of the cases. Gastro-intestinal bleeding was noted in cases of gastric and rectal carcinoids in the above series but was not observed in those originating in the ileum. In our case bleeding was the presenting sign.

Postoperatively the patient did well. The serotonin level was normal. No signs of an elevated serotonin level had been noted preoperatively. Stools were negative for occult blood and the hematocrit and hemoglobin stabilized at a normal level.

SUMMARY AND CONCLUSION

1. In any large series of cases of gastro-intestinal bleeding studied one is expected to find obscure causes 15-25% of the time.

2. If abdominal exploration is resorted to in patients with hidden causes of alimentary bleeding one can expect to find nothing in one-half of the causes.

3. In those cases of bleeding where a diagnosis is

eventually established, primary malignancy will be found 10% of the time.

4. Three cases of rare and obscure causes of alimentary bleeding have been presented and discussed.

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ABDOMINAL WOUND DISRUPTION, ITS INCIDENCE, PREVENTION AND  
TREATMENT — *Continued from Page 107*

of wounds is accompanied by many fads and personal indulgences."

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# Tracheal Replacement With Polyethylene Tubes And Report Of Case

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AND CHRISTOPHER J. STRINGER, M.D.\*\*

Successful and adequate resection of tracheal neoplasms is impossible without providing a reconstruction of the airway.<sup>1,5,7</sup>

The uniformly fatal outcome in cases of tracheal tumors, usually with terminal strangulation, provides the incentive to discover the factors essential to successful tracheal reconstruction.

From 1953 to 1955 we became interested in this problem and devised a technique for the replacement of the thoracic trachea of dogs with semirigid polyethylene tubes.

## OPERATIVE TECHNIQUE

Under ether and oxygen endotracheal anesthesia right thoracotomy is performed, the trachea freed up; following distal transection of the trachea, anesthesia is continued via a transthoracic endotracheal tube; after partial withdrawal of the transoral endotracheal tube and completion of resection with proximal transection of the trachea, the polyethylene prosthesis is invaginated into the tracheal stump, and secured with several mattress sutures.<sup>5,10</sup> The proximal anastomosis is completed first so that the transoral tube can be advanced through the prosthesis and after removal of the transthoracic tube the distal anastomosis is finally completed and anesthesia continued via the transoral tube during completion of the procedure consisting of checks for leaks under saline and closure of the chest wall using care to obtain pulmonary re-expansion. Intrathoracic penicillin was used routinely.

## MORTALITY AND RESULTS

Following the period during which the technique was perfected, the mortality was 8%. The one mortality in the last series of twelve dogs had had a pneumonectomy along with tracheal resection and died early in the postoperative period — this dog's right lung was studied with gray-white, rubbery, umbilicated lesions which on microscopic examination proved to be alveolar cell carcinoma. This case has been registered in the Armed Forces Institute of Pathology.

The remaining dogs were bronchoscoped periodically

— the bronchial tree uniformly remained healthy, the inner wall of the prosthesis always was studded with droplets of cloudy fluid, but the airway was adequate.

The original objective of this experiment was to determine whether or not the wall, which regenerates around the prosthesis, could maintain a lumen. We had reason to believe it could because of the reports of regeneration of cartilage. However, we were disappointed to discover that whenever the prosthesis was removed bronchoscopically the regenerated wall collapsed and the animals died of strangulation.<sup>1,11</sup>

Autopsies uniformly revealed a significant fibrous wall with a smooth inner surface but microscopic examination (Fig. 1) failed to show any regeneration of cartilage or mucosa in the dense fibrous tissue.<sup>5,10</sup>

The collapse of the fibrous tissue wall, which forms around the prosthesis, and occurs after removal of the prosthesis has been reported by all who have done work on this problem.<sup>1,2,4,7,10</sup>

Furthermore, attempts to contribute to the stability of the fibrous tissue wall by using autographs,<sup>3,12</sup> homografts,<sup>3,6,9</sup> or tantalum mesh wrapped around the polyethylene prosthesis resulted<sup>14</sup> in tracheal stenosis.

Therefore, we concluded that the successful use of a prosthesis demanded it be left in place permanently<sup>11,12</sup> and that there is no safe period for removal of the tube.

The last dog was sacrificed approximately one year after implanting a prosthesis in order to present evidence in favor of treating a human case of tracheal carcinoma to be reported below. It is interesting that this dog, a black, cocker spaniel, was used by a laboratory technician for upland game hunting a short time before his sacrifice.

Autopsy revealed the prosthesis in place surrounded by a thick wall of dense fibrous tissue with adequate airway and no evidence of pulmonary infection, (Fig. II).

We were further encouraged to use this method in a human by Dr. Jarvis' report of a human case of resection of a tracheal cylindroma and replacement with a stainless steel tube with a 33 months survival.<sup>8</sup> This continues to be the most successful case reported to date.

## CASE REPORT

A 52 year old white male, was first admitted to the Ingham Chest Hospital on 1/9/55 because of hemopty-

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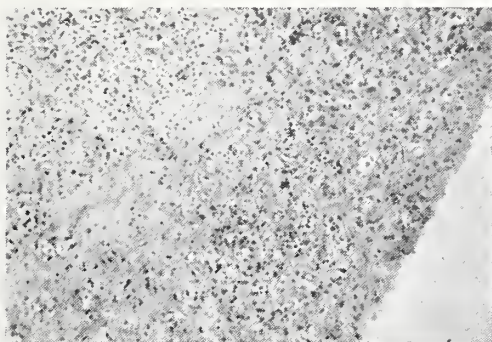


FIG. I



FIG. II

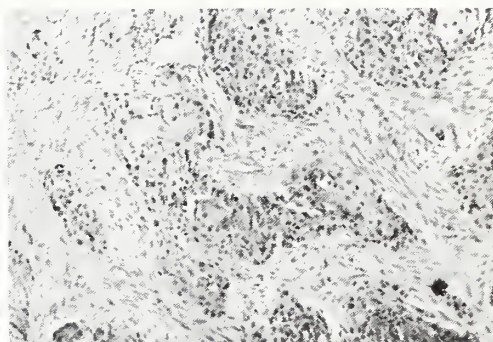


FIG. III

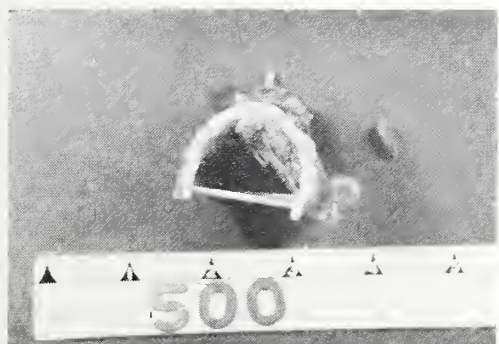


FIG. IV

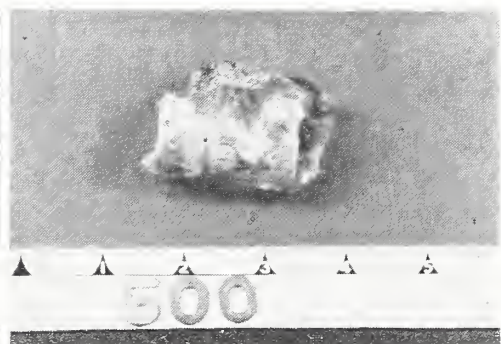


FIG. V

FIG. I. Fibrous tissue wall without evidence of epithelial or cartilaginous regeneration.

FIG. II. Trachea of dog one year after resection and replacement with prosthesis.

FIG. III. Identical microscopic picture of carcinoma of trachea and pulmonary lesion.

FIGS. IV & V. End and lateral view of resected carcinoma of trachea (8-9 tracheal rings — 7 cm. segment before retraction).

sis and a cavitory lesion of the superior segment of the right lower lobe. Bronchoscopy was reported as negative. In spite of the antibiotics, hemoptysis continued and no change was observed on x-ray. Therefore, a superior segmentectomy was done, frozen section revealed carcinoma, so a right lower lobectomy with lymph node dissection was performed. He was discharged 15 days later with a diagnosis of bronchogenic carcinoma of right lower lobe with negative lymph nodes. He was re-admitted approximately one month later with extreme stridor which began with slight wheezing 2 weeks earlier. Bronchoscopy revealed almost complete obstruction of the trachea in the mid-portion by a fleshy tumor. An adequate airway was obtained by removing the material with a biopsy forceps. Microscopic examination revealed a tumor identical to that of the right lower lobe, (Fig. III).

After due consideration of this man's desperate plight and a review of our experience with tracheal resection in dogs, resection of the tumor and permanent prosthesis was elected. This was done through a lower cervical and sternal splitting exposure with removal of paratracheal nodes and tracheotomy, otherwise the procedure was as described above for dogs. Approximately 2 cm. of tracheal stump below the cricoid and above the carina remained for anastomosis with the polyethylene tube, (Fig. IV & V).

The postoperative period was entirely uneventful until the 11th postoperative day when a large hemorrhage occurred through and around the tracheotomy tube and the patient expired in a few seconds. Autopsy revealed: No residual tumor and no source of the massive hemorrhage.

#### DISCUSSION

Dr. Lindskog<sup>15</sup> and others have indicated experience with fatal hemorrhage as a complication of tracheal replacement with a prosthesis. Others<sup>11,13</sup> have reported it. Although we were unable to demonstrate the site of hemorrhage, it was almost certainly due to erosion of a vessel by the rigid prosthesis and this factor is of utmost importance to the problem of tracheal replacement.

Dr. W. E. Burnett<sup>2</sup> in 1951 suggested that wire mesh could be wrapped around the prosthesis and become enmeshed in the fibrous tissue envelope so that it would not collapse after removal of the prosthesis. Undoubtedly this is worthy of further investigation, however, abnormal fibroplasia of unepithelialized wall with resulting stenosis is not obviated<sup>12</sup> and certainly the danger of erosion of blood vessels by the wire would be even greater.

The successful use in dogs of permanent teflon prosthesis which are relatively less rigid would warrant its trial in humans although one animal died on the 24th postoperative day of massive hemoptysis. The site of hemorrhage was not found at autopsy.<sup>11</sup>

#### SUMMARY AND CONCLUSIONS

1. Prostheses are necessary for adequate tracheal resection.
2. Collapse of the fibrous tissue wall, which forms around the prosthesis, and stenosis due to abnormal fibroplasia obviate the use of temporary prostheses.
3. The use of wire to prevent collapse of the fibrous tissue envelope does not obviate stenosis from fibroplasia and increase the hazard of hemorrhage.

4. Permanency of the prosthesis is essential.
5. Postoperative hemorrhage due to erosion of vessels by the prosthesis is a hazard with all techniques and materials.
6. A case of alveolar cell carcinoma in a dog has been reported and registered with the Armed Forces Institute of Pathology.
7. An unusual case of tracheal carcinoma with bronchogenic metastasis treated by separate pulmonary and tracheal resection with prosthesis and death from hemorrhage on the 11th postoperative day is reported.

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## View Box

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### Retropharyngeal Hematoma Caused By Vertebral Fracture

A forty-three year old man's rheumatoid arthritis was followed for the last twenty-five years. Most of his troubles were related to a Marie-Strumpell type ankylosing spondylitis which caused pain and complete rigidity of the spine and of both hip joints. He could rotate his head, as the atlanto-axial articulation were not affected, but the rest of the cervical spine and also the dorsal and lumbar spine did not show distinction of the intervertebral joint spaces. The calcified ligaments plastered the articular processes and neural arches to an uninterrupted solid block from the axis down to the sacrum.

The patient fell and hit himself occasionally, being unable to prevent himself from slipping, or protect himself when losing his balance.

Hours before admission he fell again flat on his face.

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He managed to get up by himself and did not feel any sharp pain. However, in the next hour his neck started to swell and within three hours it acquired about the diameter of his head. He could not swallow and had increased difficulty in breathing.

The lateral neck film (Fig. 1) showed a fracture line in both articular processes of C5-6 extending into the body of C5, but no compression or dislocation was present. The hypopharynx and esophagus were completely compressed. The larynx and upper trachea were pushed markedly anteriorly. The retrotracheal space measured 5cms instead of the usual 1½cms or so.

The story of the patient and the fracture suggested a hematoma presumably supplied by a torn vertebral artery or some other artery.

The patient's hemoglobin fell to 9.4 from the previous month's 12.6 figure which indicated a blood loss



FIG. 1

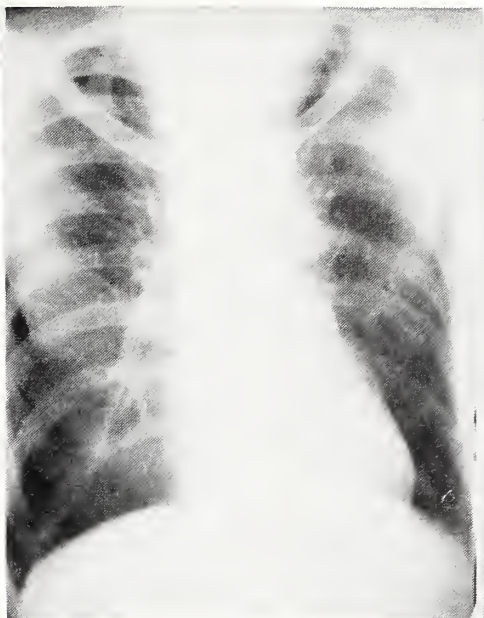


FIG. 2



FIG. 3

of approximately 1300cc into the retropharyngeal tissues. The PA chest film showed a widening of the upper mediastinum, which indicated a downward dissection of the hematoma of the pre-spinal tissues. (Fig. 2) Part of the head is projected over the upper thorax, as the patient was unable to lift it.

A tracheostomy tube was inserted and an attempt

was made to decompress the hematoma by puncture, but without success as the blood was already clotted. Infusion of fluids and transfusion was instituted.

In the next few days the hematoma was completely resorbed and the neck returned to normal size and outline. The tracheostomy tube was removed six days after admission.

## Constrictive Lesion Of Colon

A fifty-eight year old woman was admitted to the hospital because of an eight pound weight loss in two months in spite of having a good appetite. She also complained of abdominal distress and a "blown-up" feeling. Her bowel movements were getting somewhat irregular, several loose stools per day interchanging with constipation.

The past history revealed several years treatment for cavitary pulmonary tuberculosis, which ended with thoracoplasty of the right side fifteen years before this admission. Since that time she felt well, did not raise sputum and yearly check-up films did not reveal any changes on the fibrotic right side, or any disease on the normal left side. She also had a complete left bundle branch block for which she was treated at home and in the hospital with digitalis. Hemoglobin was 13.3 and urinalysis was negative. She had no fever.

Barium Enema showed an annular constriction in the mid transverse colon with a lumen of only 3mms. The rest of the colon looked normal and the cecum was also normal. The annular narrowing did not change during the course of the examination and its walls were rigid. (Fig. 3).

An operation was performed with the diagnosis of constricting carcinoma of the colon. At resection no serosal implants were seen in the surrounding tissues

and the liver and the other internal organs also looked normal.

After opening of the specimen, the adjacent wall near to the stricture was found pliable and the center of the lesion showed an unusually flat ulcer, with flat edges and no evidence of "shelving." The adjacent lymph nodes were found negative. On microscopic examination many lymphocytes were found infiltrating the entire wall and numerous granulomatous lesions were found surrounded by giant cells having peripheral nuclei. Acid-fast stain disclosed tubercle bacilli in the granulomatous lesion of the intestine.

The case was thought to be remarkable, as no tuberculous manifestations could be found in the region of the cecum, where it is common, and no diarrhea spasm or tenderness had drawn attention to the disease. The only reaction, apparently, was that of granulation induced by the specific ulceration, which occurred when the tb of the lung had been considered stable for a long time.

This case proves the well known point, that even cases which look so typical radiologically and surgically can turn out to be quite atypical.

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# The Value Of The Ear Oximeter

## In Medicine Today

FRANCIS X. MACK, M.D.\*

Oxygen has long been recognized as an absolute necessity, essential to human life. It has been well established that there is no real storage of oxygen in the body, and what little stores there are, may be rapidly depleted once the body is totally deprived of this gas. The medical literature abounds with material on hypoxia and anoxia and its detrimental effects on the human body. It is not the purpose of this paper to delve into such deleterious activity, but simply to describe the method of oximetry employed at the Mercy Hospital for the past two years, to emphasize some of the uses and advantages of such determinations in the patient with a cardiac and/or respiratory problem, and to document two interesting cases where the ear oximeter was employed and proved to be of definite practical advantage.

Oximetry simply means the measurement of oxygen, but commonly regarded today as the measurement of oxygen in the blood — either in percentage of saturation, in millimeters of Hg pressure tension, or directly in volumes percent. The three methods commonly employed today for determination of oxygenation are usually:

1. Van Slyke Method (Manometric)
2. Photoelectric Method
  - a) Ear Oximeter
  - b) Cuvette Oximeter
3. Polarographic Method

At the Mercy Hospital during the past two years we have employed the ear oximeter in determining the oxygen saturation of patients afflicted with cardiac and/or pulmonary problems and have found it to be extremely advantageous. The reasons for this opinion will be further outlined in this paper.

### THE EAR OXIMETER

In 1949, E. H. Wood published an article entitled "A Single Scale Absolute Reading Ear Oximeter"<sup>1</sup> and later another article on the "Photoelectric Determination of Arterial Oxygen Saturation in Man".<sup>2</sup> Since these articles have appeared, a modification of this instrument has been manufactured known as a Waters X60A ear oximeter which has been and is today employed in many leading hospitals throughout the country. The unit as described below consists of:

1. Specially designed multiple mirror galvanometer within suitable control assembly box and power supply.

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2. An ear piece assembly consisting of a photocell for the determination of oxygen saturation of the arterial blood in the heat flushed pinna of the intact ear and cables.

With such apparatus, it is a simple procedure for the operator to adjust the instrument to a zero point, then by the manipulation of only two dials a direct recording of the percentage saturation of hemoglobin present in the transilluminated tissue is made on the scale and read. A similar apparatus may be obtained with a direct writer<sup>3</sup> described as a Photocon Oxyhemograph Model Ox101 by Dornette and Brechner.

Measurement of the arterial concentration by the Van Slyke Method necessarily requires the drawing of blood samples at intervals which proves time consuming and somewhat cumbersome, as does the polarographic method as described by Heller and Watson.<sup>4,5,6</sup> While it may be true that a more dynamic picture of arterial oxygenation is accomplished by oxygen tension values rather than by hemoglobin saturation; for all practical purposes in the hospitalized patient, we have found that the saturation expressed as such by the oximeter currently in use has suited our need and aided in the direction of the treatment for the patient.

Arterial oxygen levels while breathing room air normally provides 97.5% hemoglobin saturation which may be interpolated into oxygen tension by the use of the oxygen dissociation curve for human blood as seen in figure one.

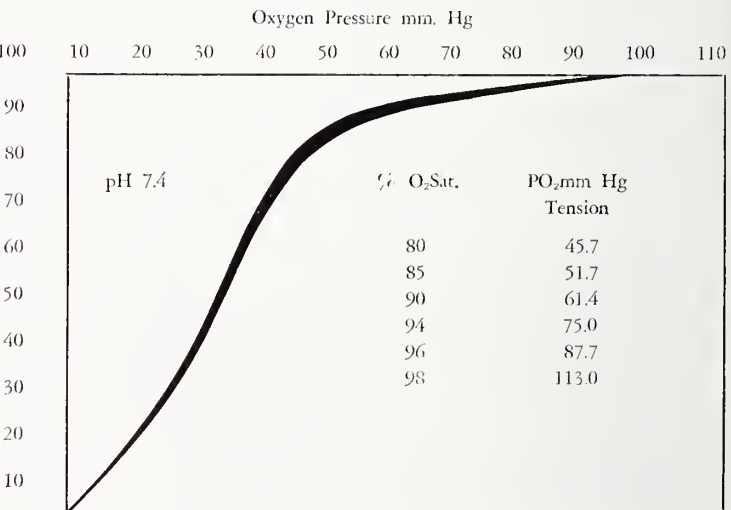


FIG. 1. Oxygen Dissociation Curve From Human Blood<sup>7</sup>

We are well aware of the fact that the oxygen saturation curve differs a bit in the presence of abnormal pH

or temperature changes but this does not alter the main concept that all patients suffering from hypoxia regardless of any such changes should receive optimal therapy to combat the oxygen deficit.

Some of the advantages of this technique employed for the determining of oxygen percent saturation include the following:

1. The instrument is readily mobile; that is, it may be transported easily to the patient whether he be in the operating room, recovery room, special care unit, or in a hospital room.
2. Determinations may be made only after a five to ten minute "warming up" period of the oximeter without inconvenience to the patient such as inserting needles or catheters into the patient's vascular system which require some dexterity, especially in those patients with poor or organically diseased arteries.
3. Continuous direct recordings are available, and changes in saturation occurring within 15-20 seconds are readily apparent while administering various concentrations of oxygen by different techniques; during anesthesia or while asking the patient to perform various tasks which might influence change of oxygen saturation of the blood.
4. Very simple and very few adjustments are required during the determinations and these may be performed by one person.
5. The instrument is compact with no additional equipment necessary except as described above, utilizing 110-115 volt line 50-60 cycle alternating current.
6. The error is extremely small, representing 2% error of the Van Slyke technique throughout the scale.

USES OF OXIMETRY IN A COMMUNITY HOSPITAL

Much of the work heretofore published dealing with oximetry has been in the realm of anesthesiology; such as, its use during bronchoscopic procedures,<sup>8</sup> oxygen inhalation prior to anesthesia with nitrous oxide or other anesthetic agents, and effects of apnea and endotracheal suction.<sup>9</sup> Reports also indicate its usage in patients with congenital heart disease<sup>10,11</sup> and during different types of anesthesia,<sup>12,13</sup> and as a circulatory monitor.<sup>14</sup>

Little work has been reported on the use of oximetry in the special care unit or in the recovery room of the hospital where it would seem that it would provide its greatest benefit. We are particularly thinking of those patients requiring treatment for bronchial asthma, emphysema, bronchiectasis, severe pneumonitis, or any other condition which apparently is causing hypoxia. It seems that it would be a relatively simple procedure to test the efficacy of not only the type of oxygen treatment being used for that particular patient, but also the efficiency of bronchodilator drugs which help reduce obstructive phenomena in the lungs themselves. The patients of particular interest might be those requiring special procedures, such as a tracheotomy for proper oxygenation and the relief of respiratory acidosis.

CASE NO. 1

Recently one interesting case was that of a fifty-five year old white male who was admitted to the Mercy Hospital with a chief complaint of increasing difficulty in breathing for the past several days.

Present illness reveals that three weeks ago this patient cleaned some marine engines with CCl<sub>4</sub> and since then had noticed he had increasing difficulty in breathing. At first he thought he had a cold, and he summoned is L.M.D. who prescribed antibiotics and bed rest. This treatment did not improve his condition and he was admitted to the hospital with wheezing respirations and difficulty in "getting his breath."

Past history revealed no previous history of asthma, pneumonia, or other respiratory or cardiovascular difficulties.

Physical examination revealed a well-developed and well-nourished white male patient in acute respiratory distress with severe apprehension. His temperature was 99°F. Head: E.E.N.T. negative, except for slight cyanotic tinge to the lips. Chest: *Heart* sounds of good quality, rate and rhythm — no murmurs. Rate 85, blood pressure 130/85. *Lungs* Expiratory wheezes throughout both lung fields. No definite râles, few rhonchi throughout, slight emphysema. Patient was using accessory muscles of respiration. Abdomen: Negative. Extremities: Negative, except for slight cyanosis of nail beds. Diagnosis: Status asthmaticus, question of chemical in origin.

This patient was treated with Chloromycetin®, Teldrin®, Adrenaline, Amminophylline, Isuprel®, oxygen, but clinically to no avail. On the third hospital day an oximetry test provided the following results:

400 CC TIDAL VOLUME — O <sub>2</sub> SATURATION			
	Oxygen at	Oxygen	Oxygen.
Breathing	6 l./m with	with mask	Positive pressure
room air	nasal catheter	at 8mm of Hg	
82%	90%	91%	92%

It is easily seen that despite oxygen therapy this patient was still hypoxic to some degree. Following the inhalation of 1/2 cc of Isuprel there appeared to be no change in oxygen saturation as the figures below indicate:

FOLLOWING ISUPREL INHALATIONS			
	Oxygen at	Oxygen	Oxygen.
Breathing	6 l./m with	with mask	Positive pressure
room air	nasal catheter		
81%	90%	90%	91%

Similar readings were obvious after Adrenaline and Amminophylline administrations. On the third hospital day, because of the persistently low oxygen saturation, it was decided to administer Medral®, 15 mg. three times daily, intravenously. The second day of treatment with Medral, the oxygen saturation figures improved and continued to do so for the next few days as the following chart indicates:

FOLLOWING MEDRAL TREATMENT

	Breathing room air	Oxygen at 6 l./m with nasal catheter	Oxygen, positive pressure
First Day:	85%	96%	97%
Second Day:	96%	98%	98%
Third Day:	96%	98%	98%
Fourth Day:	96%	98%	98%

On the eighth hospital day oxygen per nasal catheter was discontinued, the Medral was given in decreasing increments, the patient made an uneventful recovery and was discharged home on the twelfth day after admission.

COMMENT ON CASE NO. 1

We have shown by the oximetry readings that this patient's hypoxia was not benefited to any appreciable degree by the use of any one of the three commonly employed drugs: Adrenaline, Amminophylline, or Isuprel®. It was demonstrated that at least oxygen per nasal catheter elevated his oxygen saturation from 81 to 90%, but still some degree of hypoxia was existent. It was not until steroid treatment was started that his patient's oxygen saturation approached the normal of 96-98% — an almost dramatic improvement within forty-eight hours of the specific treatment.

CASE NO. 2

A seventy-two year old white male entered the hospital with "shortness of breath" and "blackouts." On physical examination the significant findings included cyanosis, bilateral rales at both bases, cardiac enlargement, blood pressure 130/80, pulse 95, respirations shallow and at the rate of 30 per minute. He was treated with bed rest, antibiotics, and fluids and was placed in an oxygen tent. When we were summoned to see him, he was in a comatose state and severely hypoxic. Our treatment consisted of intubating the trachea under direct laryngoscopy and attaching the Bennett respirator to the endotracheal tube until a tracheotomy could be performed. The tracheostomy tube after its insertion was then attached to the Bennett respirator set at 8 mm Hg intermittent positive pressure, 100% oxygen cycling at 20 per minute. Within twenty-four hours the patient recovered consciousness and oximetry studies and course of illness at this time are listed below:

OXIMETRY FINDINGS — POST TRACHEOSTOMY

	Room Air	Oxygen at 6 l./m per catheter into tracheostomy tube	Bennett Respirator	Volume Tidal in cc.
First Day:	78%	85%	90%	200
Second Day:	80%	84%	90%	250
Third Day:	78%	95%	91%	300
Seventh Day:	80%	90%	90%	400
Eighth Day:	91%	90%	90%	450

It may be seen by these figures that this patient improved his oxygenation on the third day post-

tracheostomy while breathing room air from 78% to approximately 85% after oxygen by catheter was introduced into the tracheostomy tube. It is also noted that the Bennett Assister using 100% oxygen increased this patient's oxygen even further, up to 91%. This appeared through the seventh day. By the eighth day, it is seen that this patient's oxygen saturation was 90% and remained at this figure whether oxygen was added or not or whether the Bennett was in use or not. At this time it was noticed that the patient's Tidal Volume had increased from 200 cc on the first day post-tracheostomy to 450 cc. During the next two days oximetry findings were essentially the same. After the eighth day it was advised that this patient receive digitalis, the theory being that his hypoxia may be on the basis of cardiac failure. The patient was digitalized over the course of the next three days and oximetry findings on the eleventh day post-tracheostomy revealed the following:

Tidal Vol.	Room Air	Oxygen at 6 l./m per catheter into the trach. tube	Bennett Assister
450 cc.	96%	97.5%	97.5%

Following this for the next two days the patient's oxygen saturation was followed and no essential changes were noted and on the thirteenth day the tracheostomy tube was removed after demonstrating that the patient suffered no hypoxia while the tracheostomy was closed.

COMMENT ON CASE NO. 2

The oximeter in this case was useful in providing information that this patient's hypoxia was not only due to pulmonary pathology but also to some cardiovascular derangement which was corrected with digitalis. It is interesting to note also the beneficial effect of the Bennett Assister with 100% oxygen in contrast to the simple addition of oxygen to the tracheostomy tube. This difference was approximately five percentage points in saturation.

Before the tracheostomy tube was removed we determined oxygen saturations during the closure and compared it with findings prior to its closure in an effort to prevent this patient from suffering oxygen lack following removal of the tracheostomy tube.

We have found it to be of significant advantage to determine these saturation figures prior to the removal of a tracheostomy tube which was originally performed to alleviate the patient's hypoxia.

Over two hundred determinations of oxygen saturation have been performed on patients in the operating room, recovery room, special care unit and in the wards at the Mercy Hospital.

It would seem that if patients are thought to be suffering from hypoxia regardless of the cause, the oximeter would not only qualitatively determine oxygen lack, but the quantitative oxygen deficit in the blood

and help determine the more appropriate and optimal treatment that would provide for the well being of the patient.

In this day and age we are in possession of a battery of laboratory tests to help us diagnose and treat individuals and this test performed with rapidity, accuracy and little inconvenience to the patient, we believe, is a great step forward in the progress of medicine.

We are unable to determine the number of patients who are admitted daily to the hospital and oxygen by tent, catheter, or mask is ordered almost empirically. We believe the physician contemplating the use of oxygen therapy for his patient should ask himself the following questions.

1. Is my patient hypoxic and if so, to what degree?
2. How much oxygen should be administered?
3. What technique of administration would be most beneficial to this patient?
4. Over what period of time should I continue this administration of oxygen?
5. Is my patient deriving actual benefit from the oxygen therapy?
6. What is the cause of this hypoxia?
7. What other drug or drugs besides oxygen will help my patient suffering from hypoxia?
8. What further procedures may enhance the proper oxygenation of the patient?

In answering these questions the physician will definitely be aided by the use of this oximeter.

#### SUMMARY AND CONCLUSION

Laboratory tests for the ill patient today are on an ever increasing scale as ordered by the physician. We have a very rapid, relatively simple method to determine not only qualitatively but quantitatively, the degree of hypoxia in the employment of the ear oximeter. It has definite advantages in that it is extremely accurate for all practical purposes and may indicate the relative merit of the choice of therapy not only as far as oxygen is concerned, but other drugs and/or procedures which have a beneficial action upon the cardiovascular and respiratory apparatus to achieve optimal oxygenation. The causes of hypoxia are many and varied and we believe the physician will be greatly aided in the

proper treatment of his patient by the use of this photoelectric method of determination of oxygen saturation.

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# From the Secretary's Notebook

## Summary Of Proceedings, Interim Meeting, M.M.A. House Of Delegates, April 8, 1962 At Brunswick, Maine

Called to Order at 2:30 P.M. by Ralph C. Stuart, M.D., President-Elect.

Roll Call — There was a total attendance of 51, including delegates, alternate delegates, councilors, committee chairmen and guests.

Announcement of the following members appointed by the President-Elect, Dr. Stuart, to serve on the Nominating Committee in accordance with the By-Laws, Chapter IV, Section 5:

- 1st District — Robinson L. Bidwell, M.D., Portland
- 2nd District — Paul J. B. Fortier, M.D., Lewiston
- 3rd District — Merrill J. King, Jr., M.D., Rockland
- 4th District — George E. Sullivan, M.D., Fairfield
- 5th District — Russell G. Williamson, M.D., Blue Hill
- 6th District — Linus J. Stitham, M.D., Dover-Foxcroft —  
Chairman

The report of the Nominating Committee shall be first on the Order of Business for the Second Meeting of the House of Delegates on Sunday, June 17 at 3:30 P.M. at The Samoset, Rockland, Maine.

Presentation of statement of Income and Expenditures for 1961 and proposed budget for 1963 as drawn up by the Council.\* Final action on the budget will take place at the meeting of the House of Delegates in June.

A report of the A.M.A. — Blue Shield, Senior Citizen Program as outlined at a joint meeting of the Health Insurance Committee of the Maine Medical Association and Council on March 10, 1962 was presented by Mr. Richard F. Nellson, Director of Blue Shield, Associated Hospital Service of Maine, who stated that they hope to have the plan in operation by June 1.

Mr. Stephen W. Woodberry of the Associated Hospital Service presented a brief report concerning the M.M.A. Group Insurance proposed change to BSC. Information relative to this proposed change has been sent to all members of the Association with application to be filled in and returned to A.H.S.

Dr. Charles W. Capron, a member of the Committee on Recruitment, Aid and Placement, presented a report relative to a Medical Education Loan Guarantee Program to aid medical students who need financial assistance. He stated that the committee is now getting information concerning such a plan from several banks in Maine and from the Maine Bankers Association. Such a plan would mean that the medical student could borrow the money needed directly from the bank and M.M.E.F. would guarantee a portion of the loan and pay the yearly interest of approximately 5½%. Money so borrowed would be returned by the medical student after he has com-

pleted his training and established his practice. The delegates were instructed by Dr. Stuart to "carry this back to your county societies for a vote next June."

The following amendments to the By-Laws to provide for a Speaker of the House was approved:

### Chapter IV

Section 2, Add Section 2A as follows:

"A Speaker of the House of Delegates shall be elected at the annual meeting of the Association, by the House of Delegates, at a time to be designated by the Council and published in the agenda."

Section 2B: "A Vice Speaker of the House of Delegates shall be elected in the same fashion as the Speaker."

### Chapter VI

Section 2: Delete the third sentence reading "He shall act as presiding officer at all sessions of the House of Delegates and shall give a deciding vote in the case of a tie," and the last paragraph reading "The President-Elect shall appoint a Committee on Parliamentary Procedure subject to approval of the Council. Members of this Committee shall attend meetings of the House of Delegates and the General Assembly and other such meetings as requested by the President-Elect.

Add Section 4: "The Speaker shall preside at meetings of the House of Delegates and perform such duties as custom and parliamentary usage require. He may address the House of Delegates at the opening meeting of all sessions limiting his address to matters of conduct and procedure in the House. He shall have the right to vote only in the case of a tie."

Add Section 5: "The Vice Speaker shall officiate for the Speaker in the Speaker's absence or at his request. In case of death, resignation, or removal of the Speaker, the Vice Speaker shall officiate during the unexpired term."

By-Laws — Chapter VIII, Section 1 was amended to change deadline date for payment of State dues from April 1 to June 1.

A resolution submitted by Stanley C. Beckerman, M.D., Director, Division of Cancer Control, State of Maine Department of Health and Welfare, which follows, was adopted:

Whereas: The Maine Medical Association represents the medical profession of the State of Maine, and

Whereas: This Association is aware of its responsibilities toward the citizens of the State of Maine, and

Whereas: There is mounting evidence of a causal relationship between cigarette smoking and lung cancer, and

Whereas: No less a person than the Surgeon-General of the United States Public Health Service has noted this causal relationship, and

*Continued on Page 129*

\*Copy to Councilors, Delegates and Alternates with copy of this summary following Interim Meeting.

# Maine Medical Association

## Program-in-Brief — 109th Annual Session

The Samoset — Rockland, Maine

Sunday — Monday — Tuesday

June 17, 18, 19, 1962

### Sunday, June 17

10:00 A.M. First Meeting of the House of Delegates

12:30 P.M. Luncheon

3:30 P.M. Second Meeting of the House of Delegates

6:30 P.M. Dinner

Speaker: WILLIAM B. WALSH, M.D., President, The People to People Health Foundation, Inc., Washington, D.C.

Subject: **Good Ship Hope in Action**

### Monday, June 18

9:30 A.M. to 12:00 NOON

9:30 A.M. **Films on The Management of Urinary Incontinence and New Technique for Urinary Diversion**

Scientific Program

10:00 A.M. **Recognizing the Depressed Patient and His Management**

WILFRED DORFMAN, M.D., Brooklyn, New York

11:00 A.M. **Experience with Live Attenuated Measles-Virus Vaccine**

SAUL KRUGMAN, M.D., Professor and Chairman, Department of Pediatrics, New York University — Bellevue Medical Center, New York, New York

12:00 NOON to 2:00 P.M. Luncheon

2:00 P.M. to 4:00 P.M.

Scientific Program Sponsored by the Maine Chapter, American College of Surgeons

4:00 P.M. Election of President-Elect

6:30 P.M. Annual Banquet

### Tuesday, June 19

9:00 A.M. to 12:00 NOON

9:00 A.M. **Fire and Explosion Hazards in Hospitals and their Control**

GEORGE J. THOMAS, M.D., Director, Department of Anesthesiology, St. Francis General Hospital and Rehabilitation Institute, Pittsburgh, Pennsylvania

10:00 A.M. **The Unconscious Driver** — Panel Discussion

Moderator — Secretary of State PAUL A. MACDONALD, Augusta

Subjects will include: Neurosurgery, Cardiology, and Pharmacology

Participants — To be announced

12:00 NOON to 2:00 P.M. Luncheon

2:00 P.M. to 4:00 P.M.

Program Sponsored by the Maine Medico-Legal Society

Presiding, CHARLES F. BRANCH, M.D., President, Maine Medico-Legal Society

**Problem Case in a Medical Examiner System**

GEOFFREY T. MANN, M.D., Pathologist, Chief Medical Examiner, Commonwealth of Virginia, Department of Health, Richmond, Virginia

**Seminar on Administrative Problems**

Conducted by Dr. MANN

6:30 P.M. Clam Bake

## SPECIALTY GROUP MEETINGS

### Monday, June 18

2:00 P.M. to 4:00 P.M.

Maine Society of Obstetrics and Gynecology

#### **The Borderline Pelvis**

CHRISTOPHER J. DUNCAN, M.D., Brookline, Massachusetts

Maine Chapter of the American Academy of Pediatrics

Speaker: SAUL KRUGMAN, M.D., New York, New York

M.M.A. Eye Section

#### **Lacrimal Surgery**

R. M. FASANELLA, M.D., Yale University School of Medicine, New Haven, Connecticut

Film: Cataract Surgery

Maine Society of Clinical Hypnosis

#### **Hypnosis in the Conquest of Inner Space**

BERTHA RODGER, M.D., Ridgewood, New Jersey

Maine Society of Pathologists

#### **Enzymology**

FELIX WROBLEWSKI, M.D., New York, New York

Maine Thoracic Society

#### **Solitary Lung Lesion**

EARLE W. WILKINS, M.D., Associate Visiting Surgeon, Massachusetts General Hospital; Instructor in Surgery, Harvard Medical School, Boston, Massachusetts

### Tuesday, June 19

2:00 P.M. to 4:00 P.M.

Maine Society of Anesthesiologists

#### **Operating Room Safety**

GEORGE J. THOMAS, M.D., Pittsburgh, Pennsylvania

Maine Society of Internal Medicine

Program to be announced

Maine Radiological Society

Program to be announced

### Luncheon Meetings

### Tuesday, June 19

Maine Radiological Society

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## SPECIAL NOTICES

#### **Election of President-Elect**

The election of a President-Elect will take place at the General Assembly, June 18 at 4:00 P.M.

#### **Election of Councilors**

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 17 at 3:30 P.M.

Third District — Knox and Lincoln-Sagadahoc

Fourth District — Kennebec, Somerset and Waldo

#### **House of Delegates**

Included in the Order of Business for the meetings of the House of Delegates will be final action on the Budget for 1963, Standing and Special Committee Reports, Nominating Committee Report and matters presented at the Interim Meeting of the House and published in this issue of the Journal, page 120.

#### **Golf Tournament**

DANIEL R. SHIELDS, M.D., Chairman



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine  
Department of Health and Welfare

Venereal Disease Reporting

ALTA ASHLEY, M.D.\*

Much has been said in the past few years about the increase in reported syphilis. However, since at the same time there has been an increase in the effort to have cases reported on the part of public health officials in many areas, it may be fallacious to assume that the increased numbers of reported cases represent a true increase in incidence. It is, therefore, important to determine how much of the increase has been due to the increased effort to obtain reporting.

In 1957 the Health Office of District III\*\* began to query physicians whose patients were reported as having 4+ serologic reactions on sera tested at the State Diagnostic Laboratory. Such querying was continued through 1960 and into the first quarter of 1961. From 1957 through 1959, twenty-six queries by letter are on file. There were, however, some telephone contacts made which are not recorded nor was there recorded any response to the queries. It is, however, the impression that most cases queried were said to be "old treated," "old congenital" or "serological fast," with a scattering of "false positive" reactions.

In 1960 there were 7 queries by letter, all concerning positive serologies. Again there is no record of the results of these letters. However, there was a definite response to telephone calls concerning positive gonococcal smears, a technique which was first adopted during that year. Reporting of this disease increased sharply during the year and out of a total of 124 for the state 38 cases were reported from District III. Seven out of 29 cases of syphilis were from this district, as was the only case of chancroid reported in the state (allegedly acquired by a Naval Air Corps man in Africa).

Beginning in April 1961 all laboratory reports from the State Diagnostic Laboratory of gonococcus or a suspicious purulent G-U smear, or any positive serological test on blood or spinal fluid, no matter what the titre,

was followed up by letter, telephone call, personal contact with the attending physician or by all three methods combined. Pamphlets concerning this diagnosis of venereal disease and other pertinent information on VD were sent to physicians practicing in or near the district.

Records were kept only of the letters and the physician response to those letters. The following data show the effect of this type contact and of closely allied educational stimuli:

TABLE I

	Response to		Cases reported to State	
	Queries by letter	letter by report of case	District III	State-wide
Gonorrhea	50	40	44	61
Syphilis	29	8*	6	14
Not stated	10	no record		

\*Probably represents reporting of some previously reported cases

It is not possible from this data to say whether or not the increase in reported cases of VD is a true one or merely a reflection of increased epidemiologic effort on the part of public health personnel. However, over the past two years several physicians queried have stated spontaneously that the case or cases reported are the first seen by these physicians for several years.

It is, therefore, probably correct to say that while the apparent marked increase in reported cases is, in some measure, a true increase in incidence, it is chiefly due to increased awareness of VD on the part of the practicing physician as well as an increased percentage of attended cases reported. This has undoubtedly been brought about through better understanding and closer working relationships between public health personnel and the physician practicing in the area. Such effort must be continued without letup and probably should be further increased in order that VD be brought to a standstill.

Continued on Page 131

\*District Health Officer, District III  
\*\*District III comprises 5 counties (Kennebec, Lincoln, Knox, Sagadahoc, Waldo) plus Fairfield, Smithfield (Somerset County) and Brunswick and Harpswell (Cumberland County). 1960 census 193,000

# County Society Notes

## PENOBSCOT

March 17, 1962

The monthly meeting of the Penobscot County Medical Society was held at the Tarratine Club in Bangor, Maine on March 17, 1962. There were fifty members present and the meeting was conducted by the President, Clement S. Dwyer, M.D.

Harold F. Schuknecht, M.D., Professor of Otology, Harvard Medical School, was the speaker of the evening. He gave a most interesting lecture on Otosclerosis and its treatment by the use of micro-surgical techniques in performing a Stapedectomy. He used slides and movies of great interest and provided a stimulating question and answer period.

At the business meeting a communication was read from the members of the Somerset County Medical Society which gave their opinions concerning the proposed administration of the Kerr-Mills program in the State of Maine. For a more complete understanding of the program, Mr. Frank Curran, Director of the Eastern Maine General Hospital was invited to explain the Kerr-Mills bill to the society. A discussion of the communication was held, and it was voted to file it for further information.

Richard C. Wadsworth, M.D. presented the plans of the Bangor City Health and Advisory Committee for the continuation of the present Salk vaccination program, and its suggestion to offer a county-wide oral vaccination drive in the fall of 1962. The society voted to endorse these plans.

Arthur N. Lieberman, M.D. gave a report on the activity of the latest meeting of the House of Delegates.

The society voted to send a letter of commendation to Byron V. Whitney, M.D., Chairman of the Public Relations Committee, for his outstanding work and results in organizing the series of TV programs presented by society members over station WAPI. It was felt that this represented a real effort in community service.

FREDERICK C. EMERY, M.D.  
*Secretary*

## PISCATAQUIS

March 29, 1962

A meeting of the Piscataquis County Medical Society was held at the Blethen House in Dover-Foxcroft, Maine on March 29, 1962.

After a social hour and dinner, the meeting was called to order by the President, George C. Howard, M.D.

Linus J. Stitham, M.D. was instructed to vote for a Speaker of the House of Delegates who would be a non-voting member of the Council.

Mr. Alan Bridges, a representative of the Associated Hospital Service of Maine, spoke to the society about the proposed Blue Cross and Blue Shield Plan for people over 65 years of age. After a heated discussion about the merits of the plan, the variation in benefits paid in different parts of the country despite the same premium rate, it was voted to have our representative, Dr. Stitham, vote as he sees fit in reference to the plan. The society felt as a whole that some further adjustments would be made in benefits paid by the plan.

H. Draper Warren, M.D. of Bangor brought us a message from the American and Maine Heart Funds. The fund would gladly send consultants to all hospitals to make rounds

and discuss various heart cases. Charles H. Lightbody, M.D. will be our representative at the first meeting in Bangor.

ISAAC NELSON, M.D.  
*Secretary*

## KNOX

March 6, 1962

A meeting of the Knox County Medical Association was held at the Knights of Columbus Hall in Rockland, Maine on March 6, 1962. Twenty-one members were present.

Blue Shield and other medical insurance policies were discussed at the business meeting which followed the social hour and dinner.

Ellis Dresner, M.D., Secretary of the New England Rheumatism Society from the Lemuel Shattuck Hospital in Boston, was the guest speaker. Dr. Dresner's topic was "The Management of Rheumatoid Disease and New Diagnostic Tests." He gave a very interesting lecture including presentation of unusual cases and illustrated his talk with x-rays and motion films.

April 3, 1962

The monthly meeting of the Knox County Medical Association was held on April 3, 1962 at the Knights of Columbus Hall in Rockland, Maine. Twenty-one members were present.

A social hour and dinner was followed by the regular business meeting. Edward K. Morse, M.D. gave a report of a meeting he attended recently concerning insurance for persons 65 or over. The Association was honored to have as special guest, James A. MacDougall, M.D., President of the Maine Medical Association.

Bertram Selverstone, M.D., Neurosurgeon of Boston, the guest speaker, presented a very interesting and helpful illustrated lecture on "Treatment of Cranio-Cerebro-Spinal Injuries."

MUSTAFA V. ONAT, M.D.  
*Secretary*

## HANCOCK

April 11, 1962

The meeting of the Hancock County Medical Society was held on April 11, 1962 at the Hancock House in Ellsworth, Maine.

Elizabeth E. Williamson, M.D. reported on the activities of the recent House of Delegates meeting.

The society unanimously voted to request the Secretary to submit to the Maine Medical Association copy of a letter to Dean H. Fisher, M.D., Commissioner of Health and Welfare, explaining why they do not approve of the Medical Assistance for the aged plan as proposed by Dr. Fisher. The society also requested the Secretary to again explain our stand on the Maine Medical Education Fund which we have previously expressed. The essence of this is in the following letter to the Executive Director of the Maine Medical Association:

"The Hancock County Society believes that before a large sum of money is raised for a Medical Education Fund (loan or otherwise) that the body that is to administer this fund should work out a detailed plan for the administration and use of the fund including the purposes or goals of the fund, methods

*Continued on Page 126*



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SEARLE

Research in the Service of Medicine

COUNTY SOCIETY NOTES — *Continued from Page 124*

of administration, eligibility requirements, methods for determination of monies available, amounts and methods of distribution to each accepted applicant, program for interest and capital payments by the recipients, penalties for failure to comply with conditions of the fund and establishment of the machinery to change the conditions of the fund to meet changing economic conditions.

"We believe this program should be worked out completely, presented to the House of Delegates meeting at their April meeting for later ratification by each county society and final approval at the annual House of Delegates meeting *before* the Committee on Recruitment, Aid and Placement is allowed to raise any further monies by assessments, loans or solicited gifts. The amount now raised indicates general support of the program. Professional and legal advice probably should be sought in setting up the program.

"From our point of view, we have twice been assessed, are now asked to authorize borrowing nearly one-half a million dollars, both for a program that is conceived to encourage Maine boys to go to medical school. Yet when asked by prospective students if help is available to them to go to medical school, we have to say yes, there is a program being set up but so far we have not the slightest idea how it will be set up and administered. We think the cart is in front of the horse."

Following these discussions and approval of the proposed budget for 1963, the meeting was turned over to William W. Ward, M.D. of Rockland, Maine. Dr. Ward presented a beautifully illustrated talk on "Contemporary Life in Alaska" based on his own eighteen month residence and practice in Juneau which was thoroughly enjoyed by all.

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## CUMBERLAND

April 19, 1962

A meeting of the Cumberland County Medical Society was held at the Eastland Motor Hotel in Portland, Maine on April 19, 1962. Thirty-eight members were present.

After a social hour and dinner, the meeting was called to order by the President, Robinson L. Bidwell, M.D. Jaime Goldfarb, M.D. of the Pineland Hospital and Training Center was elected to membership in the society.

Dr. Bidwell reported on the Interim Meeting of the House of Delegates and this was followed by considerable discussion of the items covered.

ALBERT ARANSON, M.D.  
*Secretary*

## KENNEBEC

April 19, 1962

A meeting of the Kennebec County Medical Association was held at the Veterans Administration Center in Togus, Maine on April 19, 1962 with thirty-three members and guests present.

Harald J. Schwarz, M.D. of Waterville was elected to membership in the society.

Ernest A. Sneddon, M.D., a member of the Department of Anesthesiology at the Massachusetts Memorial Hospital, presented a very interesting talk on the importance of blood volume studies.

EARLE M. DAVIS, M.D.  
*Secretary*

## New Members

## ANDROSCOGGIN

Basil Amfilo, M.D., 134 Russell Street, Lewiston  
John W. Friend, M.D., 49 Hampton Avenue, Auburn  
John G. Mendros, M.D., 11 Webster Street, Lewiston

## CUMBERLAND

Stanley G. Dienst, M.D., 131 Chadwick Street, Portland  
Clement A. Hiebert, M.D., 18 Bramhall Street, Portland  
Kathleen M. A. Millard, M.D., Windham Center Road, Windham

## KNOX

Albert L. Hunter, M.D., Knox County General Hospital, Rockland

## PENOBSCOT

Preston A. McLean, M.D., 209 State Street, Bangor

## YORK

Germain A. Binette, M.D., Portland Road, Saco

## Deceased

Allan R. Cunningham, M.D., 11 Everett Street, Winchester, Massachusetts, March 17, 1962

## Change of Address

## ANDROSCOGGIN

Joelle C. Hiebert, M.D.  
From — 369 Main Street, Lewiston  
To — Box 148, Norway  
Horacio A. Lichter, M.D.  
From — Central Maine General Hospital, Lewiston  
To — 54 Pine Street, Lewiston

## AROOSTOOK

Thomas G. Harvey, M.D.  
From — 46 So. Main Street, Caribou  
To — 59 Mayo Street, Caribou

## CUMBERLAND

Henry A. Hudson, M.D.  
From — 11 Gage Street, Bridgton  
To — R.F.D. #1, West Bridgton  
Howard R. Ives, M.D.  
From — 31 Deering Street, Portland  
To — 131 Chadwick Street, Portland  
DeForest Weeks, M.D.  
From — 158 Pleasant Avenue, Portland  
To — 1 Lantern Lane, Cumberland Foreside

## KENNEBEC

George Papadopoulos, M.D.  
From — Connecticut State Hospital, Middletown, Connecticut  
To — Provincial Mental Hospital, Essondale, B.C., Canada

## KNOX

Frederick C. Dennison, M.D.  
From — 52 Main Street, Thomaston  
To — 183 Main Street, Thomaston

## Necrologies

LAURA B. STICKNEY, M.D.

1879-1961

Laura B. Stickney, M.D., 81, of Saco, Maine, one of Maine's first women physicians, died on May 4, 1961.

Dr. Stickney was born in Porter, Maine on September 8, 1879, the daughter of James Anderson and Dora Fox Black. She attended Brewster Academy in Wolfeboro, New Hampshire and received her medical degree from the Boston College of Physicians and Surgeons in 1904.

Dr. Stickney had been a physician in Saco and neighboring Biddeford more than 56 years. She was honored in 1954 by citizens of Saco, who cited "Our Angel of Mercy in recognition of 50 years of faithful service."

She was an Honorary member of the Maine Medical Association and the York County Medical Society, having re-

ceived a 50-year medal in 1954 and a 55-year pin in 1959. She was also a member of the American Medical Women's Association, American Academy of General Practitioners, Parsonsfield-Porter Historical Society and the Saco Lodge of Rebekahs.

Dr. Stickney was owner of the Trull Hospital in Biddeford buying the interests of the late Dr. Paul S. Hill from his widow in 1949. She and Dr. Hill had operated the institution jointly from 1923 until the latter's death in the 1940's.

Dr. Stickney is survived by a son, Richard F. Stickney of Saco; a daughter, Mrs. John Cleary of Biddeford; a sister, Mrs. Frank C. Gevalt, Sr. of New York City; twelve grandchildren, two nephews and a niece.

JOHN B. VALENTINE, M.D.

1892-1961

John B. Valentine, M.D. of Dover-Foxcroft, Maine died on May 6, 1961 following a long illness. He was born in Ottawa, Canada on October 2, 1892, the son of Lawrence F. and Anna (Nicolini) Valentine. Dr. Valentine was a graduate of McGill University Medical School in 1921.

Dr. Valentine practiced medicine in Dover-Foxcroft from 1939 until 1947. He served at the Veterans Administration in Togus from 1947 until his return to Dover-Foxcroft in

1957, where he practiced until his death. During World War I, he served in the Canadian Army.

Dr. Valentine was a member of the Maine Medical Association, American Medical Association and the Piscataquis County Medical Society.

He is survived by one daughter, Mrs. Anne Hansen of Augusta.

RALPH E. WILLIAMS, M.D.

1908-1961

Ralph E. Williams, M.D. of Freeport, Maine died suddenly on June 23, 1961.

Dr. Williams was born in Sidney, Maine on June 17, 1908, son of the late Dr. and Mrs. Edmund P. Williams. He graduated from Oak Grove Seminary in 1925, Bowdoin College in 1929 and received his medical degree from the University of Vermont Medical School in 1935. He interned at Waterbury Hospital in Connecticut and practiced briefly in New

Gloucester before coming to Freeport, where he practiced medicine until his death.

During World War II, he served as a medical officer with the Maine General Hospital unit. He was a member of the Maine Medical Association, American Medical Association, Cumberland County Medical Association and was on the Staff of the Brunswick Regional Memorial Hospital.

Surviving are his widow and three children.

## News, Notes and Announcements

**Annual Thayer Hospital Cancer Seminar**  
**May 23, 1962**  
**Givens Auditorium, Colby College**  
**Waterville, Maine**

### PROGRAM

9:00 A.M. Registration  
 10:00 A.M. Welcome  
 MR. WILLIAM A. MACOMBER, Director of Adult Education  
 Extension, Colby College, Waterville, Maine

10:15 A.M. Carcinoma of the Lung  
 JOHN L. POOL, M.D., Associate Attending Surgeon, Thoracic  
 Service, Memorial Hospital, New York, New York  
 11:00 A.M. Management of Operable Breast Cancer  
 JEROME A. URBAN, M.D., Associate Attending Surgeon,  
 Breast Service, Memorial Hospital, New York, New York  
 11:45 A.M. Management of Tumors of the Digestive Tract  
 GORDON MCNEER, M.D., Attending Surgeon and Chief,  
 Gastric and Mixed Tumor Service, Memorial Hospital, New  
 York, New York  
 12:30 P.M. Luncheon — Roberts Union, Colby College

- 2:00 P.M. Cancer Detection in Office Practice  
EMERSON DAY, M.D., Attending Physician and Chief of Department of Preventive Medicine, Memorial Hospital, New York, New York
- 2:45 P.M. Carcinoma of the Cervix  
WILLIAM W. DANIEL, M.D., Associate Attending Surgeon, Gynecological Service, Memorial Hospital, New York, New York
- 3:30 P.M. Cancer Chemotherapy  
GEORGE C. ESCHER, M.D., Associate Attending Physician, Medical Service, Memorial Hospital, New York, New York
- 4:15 P.M. Panel Discussion — Current Problems in Cancer  
ALL PARTICIPANTS

### Mental Health Clinics

Dr. Margaret R. Simpson, Psychiatric Consultant, Division of Mental Health announces that after July 1, 1962, the Division of Mental Health will restrict its intake to referrals from other divisions within the Health and Welfare Department. This will apply to all present clinics conducted by the Division of Mental Health.

Persons interested in obtaining Mental Health Clinic Services may contact the Department of Mental Health and Corrections, Bureau of Mental Health, State House, Augusta, for information regarding local community services.

### State of Maine Board of Registration of Medicine Secretary — Daniel F. Hanley, M. D., Brunswick, Maine

#### Physicians Licensed to Practice Medicine and Surgery in the State of Maine March 13-15, 1962

##### THROUGH EXAMINATION

- Austiclinio de Abreu, Jr., M.D., Maine Medical Center, Portland, Maine
- Lutfi Y. Bayazit, M.D., St. Alexis Hospital, Cleveland, Ohio
- George M. Berberian, M.D., Georgetown Medical Center, Washington, D. C.
- Kamilo Biscevic, M.D., Good Samaritan Hospital, Cincinnati, Ohio
- Winton Briggs, M.D., Maine Medical Center, Portland, Maine
- Antonio E. Blois Castro, M.D., Lutheran Medical Center, New York
- Paulino So Chan, M.D., 18101 Lorain Avenue, Cleveland, Ohio
- Leo Cok, M.D., P. O. Box 5, Howard, Rhode Island
- James T. Duhig, M.D., New England Deaconess Hospital, Boston, Massachusetts
- Bayzar Erkman, M.D., Kansas University Medical Center, Kansas City, Kansas
- Ali Farpour, M.D., Metropolitan Hospital, New York, New York
- Cyrus Farrehi, M.D., Wayne General Hospital, Eloise, Michigan
- Max Frei, M.D., Beverly Hospital, Beverly, Massachusetts
- Antonio Isaias German, M.D., 1911 Baseline Road, Ottawa, Canada
- Hashem Gidfar, M.D., Fall River General Hospital, Fall River, Massachusetts
- Eugene J. Gorayeb, M.D., C. P. 230, Grand Falls, New Brunswick
- Gilbert R. Grimes, M.D., 4 Bayberry Lane, Lewiston, Maine
- Peter Hans Holz, M.D., 5 Kinsman Lane, Greenwich, Connecticut
- Niyazi H. Isil, M.D., 82 Warren Road, Toronto, Canada

- Yves Jerome, M.D., Boston City Hospital, Boston, Massachusetts
- John C. Karamanos, M.D., Rhode Island Medical Center, Howard, Rhode Island
- Takeo Kawamura, M.D., 6921 Nansen Street, Forest Hills, New York
- Marvin Stuart Kerzner, M.D., 362 Academy Avenue, Providence, Rhode Island
- Mojtaba Kia-Nouri, M.D., 700 North 63rd, Philadelphia, Pennsylvania
- Bulent I. Kirimli, M.D., 1564 Dwight Street, Holyoke, Massachusetts
- Ian D. MacLeod, M.D., 3 Beverly Hill, Canton, Massachusetts
- Chapour Maschouf, M.D., 8 Oakland Place, Worcester, Massachusetts
- Anna M. Dalnoki Miklos, M.D., Cushing Hospital, Framingham, Massachusetts
- Demitrios Nikolaidis, M.D., Boston City Hospital, Boston, Massachusetts
- Mehdi Orandi, M.D., 1722 Central Avenue, Augusta, Georgia
- Aldo F. Pedroso, M.D., 501 E. 32nd Street, Chicago, Illinois
- Jaime Portela, M.D., 27-17 42nd Street, Long Island City, New York
- Antonio E. Ramos, M.D., 20445 Cheyenne, Detroit, Michigan
- Woon Bok Rhee, M.D., Station B, Poughkeepsie, New York
- Julio Roberto Serrano, M.D., 393 Lexington Street, Youngstown, Ohio
- Fattah E. Shahidi, M.D., 86-06 208th Street, Queens Village, New York
- Jurgen Steinke, M.D., New England Deaconess Hospital, Boston, Massachusetts
- Judith (Szasz) Szentivanyi, M.D., 770 Albion Street, Denver, Colorado
- Tjeng-Hoey Tian, M.D., Mercy Hospital, Pittsburgh, Pennsylvania
- Resat S. Tukel, M.D., 3200 Noyes Avenue, Charleston, West Virginia
- Habibollah Veladi, M.D., Fairview Park Hospital, Cleveland, Ohio
- Ervin Wolf, M.D., 7702 E. Jefferson Avenue, Detroit, Michigan
- Yong H. Yang, M.D., University of Ottawa, Ottawa, Canada
- Hyung Je Yeon, M.D., Station B, Poughkeepsie, New York
- Cecil Leon Zagury, M.D., The Delaware Hospital Inc., Wilmington, Delaware

##### THROUGH RECIPROCITY

- Nissim Benado, M.D., P. O. Box 175, West Brentwood, New York
- Carl A. Brinkman, M.D., 47 Perham Street, Farmington, Maine
- Miroslaw W. Hnatiuk, M.D., New Jersey State Hospital, Marlboro, New Jersey
- Rhys Jones, M.D., 33 Fullerton Avenue, Montclair, New Jersey
- Manfred Kanther, M.D., Box 1000, Princeton, New Jersey
- Price A. Kirkpatrick, M.D., Thayer Hospital, Waterville, Maine
- John J. Lorentz, M.D., Box 575, Kennebunkport, Maine
- Joseph Nurenberg, M.D., P.O. Box 1453, Middletown, New York
- Ottone Renzulli, M.D., 64 Strafford Avenue, Rumford, Maine
- Theodore H. Sanford, M.D., 1202 Bolton Street, Auburn, Maine
- Samuel D. Santa Rita, M.D., Wyoming General Hospital, Inc., Mullens, West Virginia
- Harald J. Schwarz, M.D., Sisters Hospital, Waterville, Maine
- Ernesto R. Soriano, M.D., Norwich State Hospital, Norwich, Connecticut
- Frederick M. Spitzhoff, M.D., Tall Timbers Drive, Princeton, New Jersey
- Minoru Wakana, M.D., R.D. 1, Box 46, Whittier Park, Kingston, New York



### Practical Aspects Of Blue Shield

One of nine medical secretarial meetings held throughout the state by the Maine Blue Shield plan in 1961.

During 1961, more than 550 dinner invitations were issued to medical secretaries by the Maine Blue Shield plan. The purpose of these dinner meetings was to further the secretaries' understanding of Blue Shield. The program consisted of a dinner, an instructional talk by Richard Nellson, Director of Blue Shield, and a question and answer period.

### INTERIM MEETING, M.M.A. HOUSE OF DELEGATES — *Continued from Page 120*

Whereas: It has been estimated that 1,000,000 of our present population of school children will die of lung cancer if present cigarette smoking trends continue

Be It Resolved: That the Maine Medical Association, aware, as it is, of its duty to alert the citizens of the State of Maine to public health hazards, wishes to acknowledge the causal relationship between cigarette smoking and lung cancer, and

Be It Further Resolved: That this Association desires to encourage the dissemination of information regarding the causal relationship between cigarette smoking and lung cancer.

Mr. John F. Kiser, Field Representative for the A.M.A., stated that the King-Anderson bill will be considered by the Ways and Means Committee around May 15 and stressed the importance of letters of opposition to the Senators and Representatives from Maine. He also urged that members contact Chambers of Commerce, Farm Bureaus and Allied Groups in their respective areas.

Re: Social Security for physicians.

In accordance with a vote of the House of Delegates at the annual meeting in June, 1961, the following members were appointed to serve on a committee "so that this matter of the stand of this Association on Social Security can be brought up before this House at the next meeting":

Charles D. McEvoy, Jr., M.D., Bangor — *Chairman*  
George J. Robertson, M.D., Waterville  
Roger J. P. Robert, M.D., Saco

A motion that this House of Delegates vote in favor of Social Security was opposed.

A motion that the Executive Director poll the membership (to determine the stand of the Association on Social Security) was approved.

(Cards calling for a YES or NO answer — signature "optional" were mailed on April 13, 1962).

Adjourned at 5:50 P.M.

## Book Reviews

**Postpartum Psychiatric Problems — By James Alexander Hamilton, M.D. Published by The C. V. Mosby Company, St. Louis, Missouri, 1962. Pp. 147. Price \$6.85.**

In an effort "to provide this physician with diagnostic criteria that will enable him to identify those patients who are likely to require specialized care," Dr. Hamilton has presented the thesis that "there is overwhelming evidence" of the uniqueness of these states and also treatment. Unfortunately,

however, this book lacks a clear cohesive approach to the problem in his effort to champion the physiological basis and treatment of these illnesses. Contrary to the current medical philosophy of approaching the patient as a whole person, his emphasis is on the symptomology; he neglects the individual.

Dr. Hamilton openly opposes any of the accepted psychological contributions to this area and in clichés suggests an absurdity to all facets of the approach. Instead of "rejection of the child," he postulates "thyroid deficiency."

In short, Dr. Hamilton offers his opinions with subjective bias, vague terminology and descriptions, obvious organic orientation, continuity only by position and with verbose and archaic redundancy and repetitiousness.

GUY N. TURCOTTE, M.D.  
Portland, Maine

**The Mechanism of Action of Water-soluble Vitamins, Ciba Foundation Study Group No. 11. Editors: A. V. S. de Reuck and Maeve O'Connor. Published by Little, Brown and Company, Boston. Pp. 120. Price \$2.50.**

This book is the latest addition to the admirable series of monographs on current medical problems sponsored by the Ciba Foundation. As the title implies, the present volume is devoted to a summary of the recent work carried out to elucidate the way in which the water-soluble vitamins operate in the biochemical milieu.

The audience the book is directed towards is the group of workers interested in the biochemistry of enzyme activity. It is definitely not a book for browsing and unless one has a substantial understanding of advanced ideas in chemical valency and kinetics, he will be hopelessly lost trying to read the book.

For the specialist in the field, it serves as an excellent, timely progress report of the past decade on the advances made in understanding the function of the water-soluble vitamins in metabolic enzyme activity.

In Chapter 1 Theroell describes his work done with inhibitors and kinetic studies on the mode of action of Liver-Alcohol Dehydrogenase.

Snell, from the University of California, summarizes the results he has obtained with pyridoxal and metal ions as a model system to indicate the mechanism for the action of the pyridoxal phosphate enzymes.

The function of folic acid in one-carbon transfer reactions has been more clearly defined by Jaenicke. The techniques he has used are tracer studies "in vivo" and model system studies. The principal function of tetrahydro folic acid is to activate certain C-1 groups such as  $\text{—CHO}$ ,  $\text{CH}_2\text{OH}$  and  $\text{CH}_3\text{—}$  involved in carbohydrate metabolism.

Breslow discusses the fine detail of the action of Thiamin pyrophosphate via a zwitter-ion form.

Finally Lynen gives a comprehensive summary of the role of biotin in  $\text{CO}_2$  fixation.

As in the other volumes of the series, each chapter ends with a transcript of the discussion that followed each lecture. In many cases these more or less off-the-cuff discussions are more stimulating than the papers themselves, and from the differences of opinion expressed the subject matter is placed in better perspective.

JEROME R. TICHY, Ph.D.  
Portland, Maine

## Letter To The Editor

Daniel F. Hanley, M.D., Editor  
The Journal of the Maine Medical Association

Dear Doctor Hanley:

It has been a great honor and privilege for me to be honored by two special issues of the Journal of the Maine Medical Association. I thank you with all my heart.

I would like to express my special gratitude and appreciation to all my medical colleagues in Maine.

Sincerely yours,  
HANS V. MAUTNER, M.D.

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DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page 123*

## New England Health Institute

With the New Hampshire State Department of Health as host, the 28th annual New England Health Institute will be held June 19, 20 and 21 at St. Anselm's College, Manchester, N. H. The Institute was held at Colby College in Waterville in 1961 under the sponsorship of the Maine State Department of Health and Welfare.

"How to Deal With People's Fears — A Community Challenge" will be the Institute theme this year with the President-elect of the American Psychiatric Association among the discussion leaders on the related subtopic, "Organizing Community Services to Meet People's Needs." Jack R. Ewalt, M.D., Professor of Psychiatry, Harvard Medical School and Director, Massachusetts Mental Health Center, Boston, Massachusetts who is the incoming president of his national specialty group will be a discussant at the Wednesday morning session, June 20.

The timely topic of "Medical Care for People — A Critical Problem of Our Age" will be discussed by Caldwell Esselstyn, M.D., Executive Director of the Rip Van Winkle Clinic, Hudson, N. Y. at the same session. The third member of the panel will be Jerome Trichter, Assistant Commissioner of Health, New York City Health Department. Franklin Foote, M.D., State Health Commissioner of Connecticut will be the moderator.

Milford Hatch, ScD, Assistant Chief Viro Pathology Unit, Communicable Disease Center, U.S. Public Health Service, Atlanta, Georgia will conduct a laboratory workshop with demonstration on the subject of Application of Fluorescent Antibody Methods to the Study of Viral Diseases at a sectional meeting Wednesday afternoon.

Other sectional sessions on current problems at the same time period include: Crisis in Housing — Re-development and Relocation — Its Impact on People, Edward Stone, Unit Director, Social Service Unit, Government Center Project, Boston and Director of Chronic Problem Families Research Project; School Drop-Outs — A Manpower Crisis, Miss Evelyn Murray, Chief of Youth Services, U. S. Employment Service, Department of Labor, Washington, D. C.; Physical Fitness — Are We Ready?, Arthur Miller, Ph.D., Professor of Educa-

tion, Boston University; Public Relations — Our Community Image, Thomas Hoare, Public Relations Consultant, Boston College; Crisis and Solutions in Public Welfare, panel discussants, Dean Fisher, M.D., Maine Commissioner of Health and Welfare and James J. Barry, Commissioner, New Hampshire State Department of Welfare.

Dr. Murray Banks, termed a psychologist with a sense of humor, is to be the dinner speaker Wednesday evening when Joseph Cannon, Rhode Island State Commissioner of Health, will be toastmaster.

"Medical Quackery" is the provocative topic of Nevis E. Cook, Director, Boston District Food and Drug Administration for the general session Thursday morning when there will be demonstrations of on-going programs. Mrs. Dorothy G. Mullen, Recreation Consultant, Hospital Section, Connecticut State Department of Health, will discuss "Reanimation — Nursing Home Patient Activity Program" and the Rev. Fr. Kenneth Murphy of Boston, who is credited with saving the lives of a number of would-be suicides will tell of his work with "Rescue Incorporated." "A Modern Picture of Fear," a summary based on modern day radiation hazards will be presented by Eric T. Clarke, Vice President for Research and Development, Technical Operations, Inc., Burlington, Mass. Alfred L. Frechette, M.D., Commissioner of Health for Massachusetts will preside at the general session.

Following a summary and critique of the three day Institute by Harold W. Demone, Jr., Executive Director, Medical Foundation, Inc., Boston, the closing session will be held with Robert Aiken, M.D., Vermont Commissioner of Health as toastmaster at the luncheon meeting. The guest speaker will be Mrs. Dexter O. Arnold, President-elect of the General Federation of Women's Clubs whose subject will be "No Ceiling on Miracles."

Miss Ruth T. Clough, Health Education Consultant for the Maine State Department of Health and Welfare who was Institute Secretary for the 1961 session, is Maine liaison person for this year's Institute. Dr. Edward W. Colby, Commissioner of the host agency, the New Hampshire State Health Department is a former City Health Officer of Portland.

## BLUE CROSS AND OTHER INSURANCES ARE ACCEPTED



ANDREW FERGUS, M.D.  
Diplomate, American Board of  
Psychiatry

Psychiatrist in charge

PHILIP BLINDER, M.D.  
Associate Psychiatrist in charge

CARL J. HEDIN, M.D.  
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# The Journal of the Maine Medical Association

Volume Fifty-Three

Brunswick, Maine, June, 1962

No. 6

## Short Term Anticoagulation Therapy In A Patient With Hereditary Hemorrhagic Telangiectasia<sup>†</sup>

PHILIP LEVIN, M.D.,\* LOUIS J. WAINER, M.D.\*\* and HERBERT L. MARTIN, M.D.\*\*\*

Sir William Osler,<sup>1</sup> in 1901, was the first to establish the true nature of hereditary hemorrhagic telangiectasia. He reviewed a number of families and described the lesions and clinical features. Subsequently, many authors have described the various manifestations of the disease. Epistaxis, gastrointestinal lesions with hematemesis or melena, pulmonary arteriovenous fistulae with hemothorax, hematuria, and menometrorrhagia have been described. Recent reviews of the literature have been contributed by Fox<sup>2</sup> and Ecker.<sup>3</sup> In general, successful measures have been limited to local therapy and blood replacement. Systemic preparations such as vitamin K, vitamin P, snake venom, estrogens, and steroids have given equivocal results. Septodermoplasty<sup>4</sup> has been reported to ameliorate epistaxis. It is the purpose of this paper to present a new case of hereditary hemorrhagic telangiectasia and to describe the indications, results, and safety of anticoagulation therapy for concomitant cerebral insufficiency.

### CASE REPORT

R. B., a 58-year-old, right-handed, white male, was admitted to the DeGoesbriand Memorial Hospital on January 28, 1961,

because of onset of fluctuating left hemiparesis, more marked in the arm than leg of four hours' duration.

Epistaxis occurred frequently during his adult life. He has been aware of small papular areas on his nose for many years. He underwent submucous resection in 1950 for a deviated septum. Bleeding occurred, but the patient did not require transfusions at that time. During his adult life he did not bruise easily nor did he bleed readily following lacerations or dental extractions. The patient had been hypertensive since 1957. A diagnosis of diabetes mellitus was made in 1959.

The patient's mother, maternal aunt and uncle, and two cousins were also subject to hereditary hemorrhagic telangiectasia. The patient's two children, ages sixteen and twenty, do not have evidence of this disease at this time.

The first episode of gastrointestinal bleeding occurred in early 1960. He was admitted to this hospital with a hemoglobin of 6.3 gm. per 100 ml. A reticulocyte count of 2% was reported. He was given three pints of O positive blood. Subsequently, a bone marrow examination was normal. He was re-admitted in October 1960, at which time his hemoglobin was again 6.3 gm. per 100 ml. An upper gastrointestinal series was suggestive of gastritis and duodenitis. Further laboratory studies included a platelet count of 228,000 per cubic millimeter and complete clot retraction at one hour. At that time the diagnosis of hereditary hemorrhagic telangiectasia was made and the patient was discharged asymptomatic following transfusion.

Physical examination on admission revealed a well-developed, overnourished male in no acute distress. The blood pressure was 170/110, pulse 70, respiration 16, temperature 97.6° F. by mouth. A left hemiparesis was present, more pronounced in the arm than the leg. Tiny erythematous papules were present about the tip of the nose. A glass slide applied to this area caused blanching and disappearance of the lesions. There were somewhat larger lesions, 1 mm. in diameter, on the lips and tongue. They were bluish-red in appearance

<sup>†</sup>From the Division of Neurology, University of Vermont College of Medicine and affiliated hospitals.

\*Assistant Resident in Neurology, University of Vermont College of Medicine and affiliated hospitals.

\*\*Clinical Associate in Medicine, University of Vermont College of Medicine.

\*\*\*Assistant Professor of Clinical Neurology, University of Vermont College of Medicine.

and did not blanch on pressure. A stellate angioma, 3 cm. in diameter, was present over the right hypothenar eminence. This faded partially on pressure. Tiny papules were present on the anterior aspect on the left side of the nasal septum. A nasal perforation was also noted. Ophthalmological examination revealed a small cluster of vessels 5 disc diameters medial to the left optic disc.

The laboratory tests revealed the following: hemoglobin 13.8 gm. per 100 ml., bleeding time 2½ minutes, clotting time 5 minutes, prothrombin time 13 sec., (100 per cent of normal.) A tourniquet test was markedly positive. The blood urea nitrogen was 17 mg. per 100 ml. The fasting blood sugar was 88 mg. per 100 ml. and the two hour post prandial blood sugar was 108 mg. per 100 ml. Lumbar puncture done on January 3 revealed an initial pressure of 230 mm. of water. No red blood cells were seen. The protein was 64 mg. per 100 ml. Because of questionable slight xanthochromia, spinal puncture was repeated the following day at which time the initial pressure was 170 mm. of water, the fluid was crystal clear and colorless and there was no xanthochromia. One red blood cell and three white blood cells per cubic millimeter were noted. The protein was 55 mg. per 100 ml. per cent. The cerebrospinal fluid serology was negative.

About six hours after admission he had regained almost full strength of his left extremities. However, the magnitude of the hemiparesis then began to vary. When most severe, his left arm was almost totally paralyzed. After forty-eight hours of hospitalization, because of fluctuation in the intensity of the left hemiparesis, a decision to anticoagulate the patient was made. Accordingly, on January 31, 1961, he was given 200 mgs. of heparin\* intramuscularly and 75 mgs. of warfarin. During hospitalization he was well controlled on 7.5 mgs. of warfarin per day. His prothrombin activity remained between 22 and 27 per cent of normal. Some fluctuations in the strength of the left extremities continued; but the remainder of his hospital course was characterized by steady improvement. By February 20 he was able to oppose his digits successfully. Ambulation was possible with the aid of a cane. Anticoagulation therapy was discontinued on February 22. He was discharged the following day. During hospitalization the patient experienced several minor episodes of epistaxis which required only small anterior vaseline nasal packs. Epistaxis was hardly more prominent than prior to anticoagulation therapy. Stool guaiac examinations were slightly positive, probably due to swallowed blood. No untoward bleeding episodes occurred. His hemoglobin remained at the same level during hospitalization.

#### DISCUSSION

The diagnostic feature of hereditary hemorrhagic telangiectasia have been described by Hanes.<sup>5</sup> They are: (1) family history, (2) repeated hemorrhages, (3) multiple telangiectasia. The family history may be easily elicited; indeed, one family has established strong traditions regarding this disease.<sup>6</sup> Often members of the same family are subject to hemorrhages involving the same system. Atavism, or skipping of a generation, has been described by some and refuted by others.

The lesions generally appear after the age of 20. Hemorrhages may occur at any age, but are more common from middle life onward. The hemorrhages may be sudden and severe, requiring multiple transfusions. Epistaxis is the most common manifestation. The mortality rate from the disease is estimated to be 4%.<sup>7</sup> Multiple telangiectases occur in all cases. Thus, in

order of frequency, they occur on face, lips, nares, tongue, ears, hands, chest, and feet. Central nervous system involvement is described by Byrd.<sup>8</sup> Pathological examination reveals thin-walled veins throughout the white matter; being particularly numerous in the subcortical area. Three types of cutaneous lesions are described by Ecker.<sup>3</sup> These are (1) pin point lesions which fade on pressure, (2) stellate angiomas, consisting of a punctiform central area from which radiate easily visible vessels; they are bluish or red in color and fade partially on pressure, (3) nodular angiomas which are rather solid looking, red or cyanotic lesions which are 1 to 3 mm. in diameter and not obliterated by pressure. It is pointed out that any site may bleed at times in the form of a sudden, arterial, spurting jet. Histologically the vascular wall is extremely thin, often consisting only of a single layer of endothelium lying immediately below the markedly thinned out epidermis.<sup>9</sup> Muscular elastic coats are absent. Because of the thin walls and the proximity to the surface, the sinuses bleed readily with slight trauma and, because of the lack of contractile elements in the wall, bleeding may not cease spontaneously.<sup>10</sup>

The vast majority of patients have no defects in their bleeding and clotting mechanisms. Some of the patients have a positive tourniquet test alone. Singer and Wolfson<sup>11</sup> report three such cases. William<sup>7</sup> reported a case with a positive tourniquet test but normal bleeding and clotting times. In this case the prothrombin consumption and the plasma recalcification time were normal. Wells<sup>12</sup> reported three cases of the disease in one family. The first patient had a positive tourniquet test, prolonged bleeding time and variable clot retraction. The second had a prolonged bleeding time, thrombocytopenia and variable clot retraction, and the third had a positive tourniquet test alone. Further study of coagulation factors was not carried out.

In the case presented, anticoagulation therapy was instituted for treatment of "a stroke in progress." The diagnosis of hereditary hemorrhagic telangiectasia was known prior to institution of anticoagulation therapy, and, on theoretical grounds, it was felt that a trial of anticoagulation therapy was safe. Heparin and warfarin were given and the patient was successfully anticoagulated. Minor epistaxis occurred; but, since this was hardly more pronounced than usual, anticoagulation was continued. During the period of anticoagulation, the patient's hemoglobin remained the same. No untoward hemorrhagic episodes occurred during the twenty-three day course of anticoagulation therapy. Anticoagulation therapy was discontinued because no further evolution of "the stroke in progress" took place.

If one accepts the thesis that the bleeding tendency in hereditary hemorrhagic telangiectasia is due to the lesions noted and that bleeding occurs when a lesion is traumatized or when it undergoes spontaneous rupture in the course of its evolution, then one need not withhold anticoagulation therapy in such cases since

\*in the form of Depo®-Heparin, Upjohn, Kalamazoo, Michigan

the anticoagulants assuredly do not affect the integrity of the lesions themselves. Were a lesion to spontaneously rupture during the course of anticoagulation therapy and major bleeding occur, it would be best to discontinue anticoagulants and to attempt local therapy in that area. To our knowledge, this occurrence on anticoagulation therapy has not been reported in the literature.

#### SUMMARY

The safety of anticoagulation therapy (heparin and warfarin) for cerebral insufficiency in a patient with hereditary hemorrhagic telangiectasia is discussed. Although this patient had gastrointestinal hemorrhage in the year prior to admission, this did not recur as a result of anticoagulation. The mechanism of anticoagulation therapy is quite apart from the hemorrhagic tendency in hereditary hemorrhagic telangiectasia, in which the thin-walled sinusoids tend to rupture easily.

Acknowledgement is made to Matthew C. Kartch, M.D., for his assistance in the ophthalmological examination of the patient.

#### REFERENCES

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2. Fox, M. I., Hereditary hemorrhagic telangiectasia. *Conn. Med.* 23:224-230, 1959.
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#### HOPE FOR NEW STEROIDAL AGENTS

During the last eight years much knowledge has been gained regarding the effects of chemical alterations on the physiologic properties of steroids. It is hoped that the pharmaceutical industry will continue to devise and test new modified steroidal compounds as it seems reasonable to expect, as more information regarding structure-function relationships is accumulated, that chemists may be able to create anti-inflammatory steroidal agents which are more efficient and safer than those that are now available. It would indeed be a severe blow to the progress of medical research if legislation is approved that would discourage continued efforts to modify the molecular structures of steroids and to impede the introduction of new drugs that may result from such effort. — Edward B. Boland, M.D., University of Southern California School of Medicine, to Senate Subcommittee on Antitrust and Monopoly.

# Health Mobilization In Total Warfare\*

CLYDE I. SWETT, M.D.\*\*

To the county medical society, health mobilization should mean active participation in bringing to a state of readiness all of the health resources of the county in close cooperation and coordination with its county civil defense health services. This not only includes its own manpower resources but also includes assisting in the coordination of all other allied professional manpower in an overall mobilization that takes into consideration every other health resource available in the county.

You have been told that the objective of such health mobilization is to help a people survive a sudden, nation-destroying catastrophe; to save as many lives as possible in the shortest time; to help rehabilitate and protect the health of the survivors; that the effectiveness of such health mobilization depends entirely upon the state of preparedness before the impact; that to be properly prepared the county medical society must study and know the assumed problem and all of its ramifying possibilities in so far as possible; make an accurate evaluation of the county's present health resources; and understand the extent of the inadequacies in meeting the problems of total warfare. Then, following this study, orientation and evaluation, the county medical society must bring itself into a state of readiness for complete health mobilization by good organization, persistent training, constant revision of its objectives, and complete coordination with and active participation in the county civil defense health services program.

I believe we all fully recognize that basically the individual physician is the key figure in any health mobilization structure of the county medical society's effort for readiness in total warfare; that the society's over-all effectiveness will be entirely dependent upon the state of readiness of each one of its individual members. For in the immediate post-impact period, it is the local physician who will have the primary responsibility for any effective medical care given. His job at such a time will be tremendous in terms of the disparity of resources and the work-load, for it will be a long time before he receives any type of organized medical support. He will need to improvise and use whatever resources are at hand. He will need to assume unfamiliar and unusual responsibilities for which he may not be adequately prepared — and the most effective time in which to save these lives is within the first two to four hours following impact.

In the immediate post-attack situation, the physician will no longer be able to work in his usual peacetime patterns. He will need to make fast decisions as to which cases to handle first, which are safe to move, which should have delayed treatment, and which are too hopelessly injured to survive. This rapid and accurate sorting becomes a must and great judgment will need to be used in making these decisions.

This disparity of personnel, supplies, equipment and medical facilities becomes a major factor in the rush for survival. His need for a previously trained health personnel in expanded function is of paramount importance to his effort. Conservation of his energy and time by good leadership and know-how in delegating much of the medical load to others becomes essential. In addition to this expansion of his manpower resources, he will need to employ uniform treatment procedures to avoid costly confusion and delays. He will need to have a good knowledge of the area for possible sites for additional medical facilities and for obtaining additional supplies.

Just as the individual physician is the key figure in all-out health mobilization, so must his county medical society become the agency through which he becomes most effective in organizing his capacities for survival medical care. It becomes the responsibility of his county medical society to help him promote sound mass casualty care planning; to provide adequate expanded medical training programs for allied professional personnel; to provide him with additional training in the techniques of disaster medicine; to plan for the complete utilization of all available health personnel resources; to promote and ensure optimum amounts of required equipment and supplies, stock-piled strategically and in a state of readiness; and to ensure prompt mobilization of all available personnel in preassigned locations. I re-emphasize that it is the responsibility and duty of the county medical society to organize, plan and then train each one of its members in the principles of disaster medicine. The sooner this is accomplished the better the conditioning of each doctor for active participation in the health mobilization effort. What consistent evidence has been shown that human nature has been changed to the extent that there will never be another war? Man with his behavior patterns still largely unchanged will still attempt to annihilate himself as in the past.

The question is frequently asked, "Specifically, just how do we go about doing this job?" In the remaining time, let us see what can be done to implement the health mobilization program I have just reviewed.

\*Paper presented at the 12th County Medical Societies Conference on Disaster Medical Care. Sponsored by Council on National Security, A.M.A. Nov. 4-5, 1961, Palmer House, Chicago.

\*\*Chief, Health-Medical Services, Maine Civil Defense.

First, however, keep in mind that any ideas I present are only meant to help give direction and to stimulate your own thoughts toward clarification of approach to the problem. You are already familiar with much of the material I am presenting but repetition also has its merit. It is important that all organizational effort and planning be kept "fluid" and subject to constant revision as experience dictates. I am also aware that what might be useful in one county situation might not be applicable to the circumstances in another county.

For the county medical society to get off to a good start, I would suggest that its Disaster Medical Care Committee be composed of certain key members of the society. The chairman of this committee could bring continuity from the state level if he were to be appointed as a member of the Disaster Medical Care Committee of the State Medical Society. Others on this committee should be the County Civil Defense Health Services Chief, who should be recommended for appointment to this civil defense position by the county medical society; the County or District Health Officer, if a member of the society; the County Medical Advisory Board Member to the State Medical Advisory Board of the State Selective Service System; and as many others as may be required to do the job. This committee has far reaching responsibilities and so should be one of the most active standing committees of the society.

The County Civil Defense Health Services Chief, the County Advisory Board Member to the State Selective Service System and the County Health Officer would act as a County Advisory Health Manpower Board to the County Civil Defense Health Services and the Advisory Health Manpower Board of the State Medical Society as to availability of physicians in the county for displacement in times of emergency, be it to give assistance in areas outside the county in a civil defense situation or for call to duty in the Armed Forces. In this way, the manpower needs of the military and civil defense agencies can be actively and effectively coordinated. This board should also work in close liaison with the county manpower office of the Department of Labor.

With respect to working relationships between the Manpower Agency (Department of Labor) and the County Civil Defense Agency, it would be well for the Society Advisory Health Manpower Board to assist in the formulation of an agreement on the responsibilities as regards health personnel listed in Annex 18, National Plan, Appendix 1, Sections 1 and 2. The County Civil Defense Health Services should be responsible for the evaluation of specific skills matched with specific persons in the classifications listed under (Hard Core) "Essential Health Skills" in a post-impact emergency. It should also say who is to be moved and who goes where with regard to persons in those skills but should have command relationship to only the essential health skills.

The Manpower Agency should provide the administrative mechanism and means to accomplish the de-

cision of the Health Agency upon receipt of the communication authorizing the move. The Health Agency will need to utilize its knowledge and records of personnel included in Sections 1 and 2, Appendix 1, Annex 18, National Health Plan (Health Manpower) for the purposes of establishing preattack planning and training.

In a post-impact emergency, the Health Agency would compete with other elements for persons within the "Supporting Health Skills" (NP-18-1) to be furnished through the Manpower Agency. The Manpower Agency would adjudicate claims and allocate these resources and arrange for the delivery of this manpower group in accordance with priorities and transportation needs established through the Civil Defense authorities.

For the county medical society to be in a state of readiness for health mobilization in total warfare, the following measures should be instituted and completed:

1. Develop a PERSONNEL ROSTER at the county level according to the health, skills, and categories as outlined in the National Plan-18-1, by name, age, address, professional qualifications and present professional assignment.
2. Submit copies of the Personnel Roster to the County Civil Defense Health Services, the State Civil Defense Health Services and the State Disaster Medical Care Committee.

Disaster Medical Care Committees of those county medical societies that include more than one county should submit copies of the Personnel Roster to each of the County Civil Defense Health Services.

3. The County Advisory Health Manpower Board of this committee provides for better coordination in displacement of physicians for the armed forces and civil defense agencies outside the county by setting up a complete AVAILABILITY ROSTER giving a tentative "availability" rating to each physician listed. Copies of this Availability Roster are submitted to the County and State Civil Defense Health Services, the State Medical Advisory Board, the Selective Service System and the State Disaster Medical Care Committee.

In the post-attack situation, to provide maximum medical care to the greatest number of casualties under the conditions of critical disparity of health manpower that will prevail, it will be imperative to commandeer and displace large numbers of physicians and allied health personnel into disaster areas.

4. From the Personnel Roster, each county committee should submit recommendations for team assignments, especially in the following hospital facilities:
  - a. Present fixed hospitals (permanent)
  - b. Relocation hospitals (auxiliary)
  - c. Civil Defense Emergency Hospitals (CDEH)
5. The county committee should organize, train and indoctrinate physicians who will assist, advise and conduct training sessions when required; to expand health personnel functions; to demonstrate their assumption of leadership in the field of disaster medicine; and to ensure adequate and proper instruction of the general public.

In a matter of seconds after impact — every available physician will become a surgeon working under tremendous mental and physical strain, with markedly inadequate supporting personnel, equipment, supplies and medical facilities. Assignments will necessarily be made to any and all kinds of the then existing and improvised hospital facilities of every description and kind following the impact. Preplanning and teaching of expanded function following the basic principles for mass casualty care are an im-

portant responsibility of the County Disaster Medical Care Committee.

6. The county committee should orient and indoctrinate itself by a thorough study of the state and county civil defense operational survival plans or annexes and be prepared to submit recommendations for revisions whenever indicated in its advisory capacity. Such recommendations on local strategic situations and operational policies can be exceedingly helpful to the efficient operation of the survival plan.
7. The committee should become familiar with charts and maps that give the locations of every hospital and other health institution in the county along with the companion lists giving the type of health facilities available, the bed capacities, the health personnel and other health resources.

Similar maps and lists or inventories should be made available that show the location and number of the mobile support units, such as the Civil Defense Emergency Hospital, Emergency Treatment Stations, organized First Aid Stations and Civil Defense Stockpiles in the county.

8. Finally, the county committee should encourage and participate in the repeated testing of the operational procedures for complete health mobilization of the county in total warfare. These actual rehearsals can best be accomplished by centering the activity around the hospitals and other existing health facilities in the county. Such tests of plans should be done at least twice a year, followed by evaluation and revision.

#### SUMMARY

In summary, the county medical society has the tremendous responsibility of planning, orientation, adaptation of health and medical function in mass disaster situations. To meet this responsibility, it must develop training objectives, training and planning participation, emergency functions of allied health personnel, triage and mass casualty treatment principles of disaster medicine.

#### REFERENCES

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4. Training of Professional Medical Personnel, U.S. Dept. of Health, Education and Welfare, Public Health Service, Division of Health Mobilization.
5. The National Plan, Annex 18, Appendix 1, Sections 1 and 2.

18 Sherman Street, Island Falls, Maine

#### CASE HISTORY WITH HAPPY ENDING

This little girl of seven had a tummy ache, but her family didn't pay much attention. When we saw her she had a ruptured appendix and a temperature of 104 degrees. We put her in the hospital and took out her appendix, sucked out some of the peritonitis resulting from it, put some medicine in, gave her medicine in the veins for a couple of days. She had no abnormal temperature after three days, then was put on medicine by mouth and left the hospital on the seventh or eighth postoperative day. The drugs must have cost \$30-40, maybe more, maybe less. But I'd like anybody to ask the parents of that youngster how much they were worth. We don't say how much does it cost but how much is it worth. —Edward R. Annis, M.D., in *Journal of Indiana State Medical Association*, July 1961.

# The Journal of the Maine Medical Association

DANIEL F. HANLEY, M.D., Brunswick, Editor

## EDITORIAL BOARD

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## Across The Desk

### Maine's Flying Physicians

Maine's doctors cover a lot of territory in the course of a day and several of them fly their own planes to catch up on the miles they have to travel. George F. Higgins, M.D. of Presque Isle flies regularly to meetings and recently carried two of his colleagues along to a meeting in Brunswick concerning the "Implementation of the Kerr-Mills Bill."

The big gathering, however, will take place when Niles L. Perkins, Jr., M.D. and Harold L. Osher, M.D., both of Portland and Paul A. Fichtner, M.D. of Bath will lead a group of Maine's flying physicians on flights to Rockland to attend the annual session of the Maine Medical Association which will be held there on June 17, 18 and 19, 1962.

### Physicians Need To Know More About Medical Technologists

What physicians and hospitals "may be getting when they hire graduates of some commercial medical technology schools" is documented in a recent issue of *The Modern Hospital*.

The report describes inadequate training and high pressure recruiting methods, excessive tuition charges, lack of qualifications among instructors and flexible standards for bestowal of degrees and certificates. It will interest physicians on the receiving lists for circulars soliciting employment for graduates of these schools, as well as physicians whose guidance is sought by young people interested in medical technology careers.

Confusion exists in the minds of many doctors be-

cause they are not as familiar with the MT(ASCP), which is the professional rating of the medical technologists, as they are with the "R.N." that distinguishes registered nurses from other nursing personnel.

The medical technologists who use the professional designation MT(ASCP) have had three years of college with a basic background of science, plus a fourth year of clinical training in a hospital school of medical technology headed by a pathologist and approved by the AMA. They have passed national boards given by the Registry of Medical Technologists of the American Society of Clinical Pathologists in Muncie, Indiana. The professional organization of this group is the American Society of Medical Technologists, the ASMT, of Houston, Texas.

The designation "M.T." is awarded commercial school graduates and others by a self-constituted agency, the American Medical Technologists, the AMT, which operates out of Enid, Oklahoma and is not listed among national accrediting agencies by the U. S. Office of Education. Neither is it recognized by the American Medical Association. Commercial of private schools of medical technology, *The Modern Hospital* article states, "are not accredited by the usual accrediting bodies for academic institutions nor recognized by such professional organizations as the American Hospital Association, the Catholic Hospital Association, the American Medical Association, or the American Society of Clinical Pathologists."

Commercial schools charge tuition fees up to \$1,395, the article states, and may have as many as 40 students

*Continued on Page 158*



DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## Poliomyelitis Prevention—1962\*

With the licensing of Type III oral poliomyelitis vaccine, the United States, through its Public Health Service, takes another major step toward the final conquest of paralytic poliomyelitis. Now two effective weapons, the formaldehyde-inactivated vaccine and the oral vaccine, are available for general use. Their proper application should accelerate the decline in poliomyelitis and could lead to the early elimination of the disease.

An Advisory Committee on the Control of Poliomyelitis was formed in the fall of 1960 for the purpose of reviewing all phases of poliomyelitis prevention. This Committee has been active since then and has prepared recommendations concerning the best use of all available weapons to control the disease.

Increased interest in the use of poliovaccines is anticipated. Since the supplies of both the formaldehyde-inactivated and the oral vaccines may be limited at this time, the present statement and recommendations should be considered as an interim document applicable to the 1962 poliomyelitis season.

The Committee on the Control of Infectious Diseases-American Academy of Pediatrics has made available a suggested outline of community organization for the use of the oral vaccine. This may be obtained by any physician on request to the Division of Maternal and Child Health, Department of Health and Welfare, State House, Augusta.

**A. Formaldehyde-Inactivated Vaccine.** This vaccine was licensed in 1955. Since then it has been used extensively in this country and in many parts of the world. In all areas where its use has been widespread, great reductions in incidence of paralytic poliomyelitis have been observed. In the United States, where over four hundred million doses have been administered, the decline in paralytic poliomyelitis has exceeded 90%. However, the vaccine has been used to varying extents in different population groups. Over 65% of preschool children and about 70% of young adults, particularly males, have not received the recommended

series of four injections. Outbreaks, and even some severe epidemics, still occur. These have been confined largely to the unimmunized or incompletely inoculated groups, but cases of the paralytic form of the disease have continued to be reported among individuals who have had three and even four doses of the vaccine.

**B. Oral Vaccines.** Research by many workers in this country and overseas during the past decade has brought this vaccine to a point where its widespread use can be recommended in the United States. Live oral vaccines for Type I and Type II were licensed in the United States in August and October 1961, respectively. Type III is now also available. These vaccines have been used in a number of field trials and community immunization programs in the United States. Methods of administration, dosage levels and factors influencing response have been determined. Effectiveness as measured by antibody response has been established. In a number of other countries oral vaccines have been used extensively, particularly where formaldehyde-inactivated vaccines have had no or only limited use. Reports have been universally favorable. Immediate and marked reductions in the incidence of the paralytic form of the disease have occurred.

**C. Relative Advantages of the Two Vaccines.** The formaldehyde-inactivated vaccine presents the advantages that stem from seven years of successful use. Its effectiveness is established. It can be combined with diphtheria, tetanus and pertussis antigens as part of routine pediatric or well-baby clinic practice. For those who have received a partial series of immunizations, a single injection calls forth a prompt antibody response to all three types of poliomyelitis. There is considerable evidence from several epidemic studies that immunization with this vaccine has induced some degree of herd immunity, although it is known that well immunized persons can become intestinal carriers of both wild and attenuated strains.

One disadvantage of the formaldehyde-inactivated vaccine is the necessity for its being injected. The cost of, and resistance to, an injection procedure may reduce acceptance by some population groups. The use

*Continued on Page 155*

\*This is the first of two articles on the general subject of Poliomyelitis Prevention. The second, Guidelines for Use of the Oral Vaccine, is scheduled for publication in the July issue of The Journal.

# Program . . .

109th Annual Session  
Maine Medical Association

Sunday - Monday - Tuesday

JUNE 17, 18, 19 — 1962

*The Samoset, Rockland, Maine*

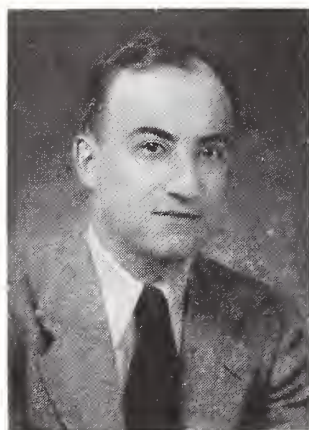
## Program

Arranged by the Scientific Committee

SIDNEY R. BRANSON, M.D., South Windham,  
Chairman

JAMES E. POULIN, M.D., Waterville

ROBERT L. OHLER, M.D., Togus



Dr. Branson

## Information

### Registration:

Registration Headquarters throughout the session will be in the Lobby at The Samoset. Registration fee \$1.00.

Sunday June 17 — 9:00 A.M. to 5:30 P.M.

Monday June 18 — 8:30 A.M. to 5:30 P.M.

Tuesday June 19 — 8:00 A.M. to 5:30 P.M.

### Visiting Delegates:

Introduction of Visiting Delegates will take place at meetings of the House of Delegates on Sunday, June 17 or at the General Assembly, Monday afternoon, June 18.

### Scientific and Educational Exhibits:

These exhibits, which are listed elsewhere in this program, will be located in the Ballroom.

### Technical Exhibits:

The technical exhibits are easily accessible to the Ballroom (where Scientific Sessions will be held), to the Dining Room and to the Golf Course. A list of these companies will be found in this program. Please show your appreciation by visiting these exhibits at every possible opportunity.

### Sponsors:

The speakers for the scientific programs are supported in part by a grant from Merck Sharp and Dohme Postgraduate Program and from Eli Lilly and Company.

## Sunday, June 17

10:00 A.M. First Meeting of the House of Delegates  
RALPH C. STUART, M.D., President-Elect presiding

12:30 P.M. Luncheon

3:30 P.M. Second Meeting of the House of Delegates  
DR. STUART, presiding

6:30 P.M. Dinner

Speaker: WILLIAM B. WALSH, M.D., President,  
The People to People Health Foundation, Inc.,  
Washington, D.C.

Subject: **Good Ship Hope in Action**



Dr. Walsh

## Monday, June 18

### Scientific Program

*Presiding* — SIDNEY R. BRANSON, M.D.

10:00 A.M. **Experience with Live Attenuated Measles — Virus Vaccine**

SAUL KRUGMAN, M.D., Professor and Chairman,  
Department of Pediatrics, New York University  
— Bellevue Medical Center, New York, New York

11:00 A.M. **Recognizing the Depressed Patient and His Management**

WILFRED DORFMAN, M.D., Clinical Instructor,  
State University Medical Department of Psychiatry,  
Brooklyn, New York

12:30 P.M. Luncheon



Dr. Krugman



Dr. Dorfman

### Scientific Program

*Presiding* — ROBERT L. OHLER, M.D.

**Sponsored by the Maine Chapter of the American College of Surgeons and the Maine Trauma Committee**

2:00 P.M. to 3:00 P.M. *Presiding*, THOMAS A. MARTIN, M.D., Portland, for the Maine Trauma Committee

#### **First Aid Treatment of the Injured Skier**

STANLEY B. COVERT, M.D., Kingfield, Maine

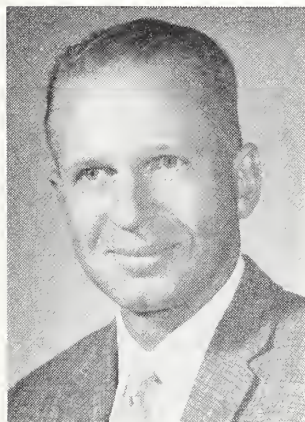
#### **The End Care of Skiing Injuries**

ARTHUR E. ELLISON, M.D., Williamstown, Massachusetts

3:00 P.M. *Presiding*, FRANCIS A. WINCHENBACH, M.D., Bath, President of the Maine Chapter of the American College of Surgeons

#### **Reconstructive Surgery of the Aorta and Large Arteries**

ROBERT R. LINTON, M.D., Brookline, Massachusetts



Dr. Covert



Dr. Linton

4:00 P.M. General Assembly

*Presiding*, JAMES A. MACDOUGALL, M.D., President

Election of President-Elect

6:00 P.M. Cocktail Party — Sponsored by Brunswick Publishing Company, Brunswick, Maine

6:30 P.M. Annual Banquet

Presentation of Honorary Pins

Speaker: Commander GEORGE P. STEELE, U.S. Navy, Staff, Deputy Commander Submarine Force, U.S. Atlantic Fleet, U.S. Naval Submarine Base New London, Groton, Connecticut

Subject: **Trans-Polar Cruise of the Nuclear Submarine Seadragon**



Commander Steele

**Tuesday, June 19**

### Scientific Program

*Presiding* — JAMES E. POULIN, M.D.

#### **9:00 A.M. Fire and Explosion Hazards in Hospitals and Their Control**

GEORGE J. THOMAS, M.D., Emeritus Professor of Section on Anesthesiology, University of Pittsburgh, School of Medicine; Director, Department of Anesthesiology, St. Francis General Hospital and Rehabilitation Institute, Pittsburgh, Pennsylvania



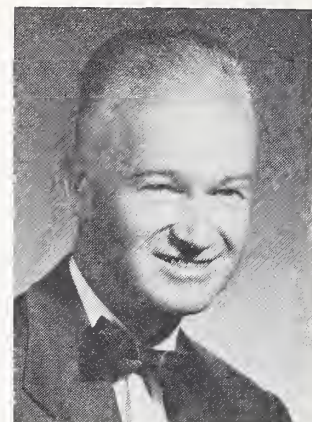
Dr. Thomas



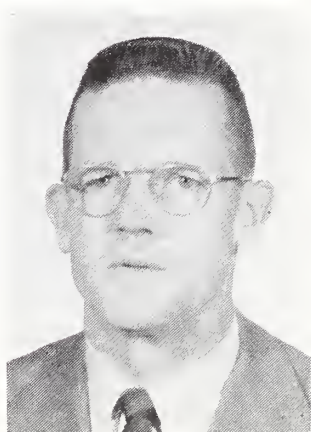
Mr. MacDonald



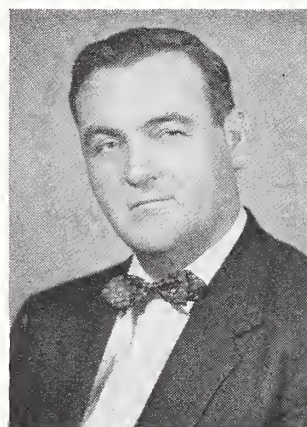
Mr. Kerrick



Dr. Chapin



Dr. Manter



Dr. Mann

**10:00 A.M. The Unconscious Driver — Panel Discussion**

Moderator — Secretary of State PAUL A. MACDONALD, Augusta

**Participants:**

MR. JOHN C. KERRICK, Director, Driver License Program American Association of Motor Vehicle Administrators, Washington, D.C.; GEORGE L. MALTBY, M.D., Neurosurgeon, Portland; WILBUR B. MANTER, M.D., Cardiologist, Bangor and MILAN A. CHAPIN, M.D., Internist, Lewiston

12:30 P.M. Luncheon

**Scientific Program**

2:00 P.M. to 4:00 P.M.

**Sponsored by the Maine Medico-Legal Society**

Presiding, CHARLES F. BRANCH, M.D., President, Maine Medico-Legal Society

**Problem Case in a Medical Examiner System**

With illustrative slides. Followed by question and answer period.

GEOFFREY T. MANN, M.D., Pathologist, Chief Medical Examiner, Commonwealth of Virginia, Department of Health, Richmond, Virginia

**Seminar on Administrative Problems**

Conducted by Dr. MANN

6:30 P.M. Clam Bake

Presentation of Golf Prizes by

DANIEL R. SHIELDS, M.D., Chairman, Golf Tournament

## Specialty Group Meetings

**Monday, June 18**

2:00 P.M. to 4:00 P.M.

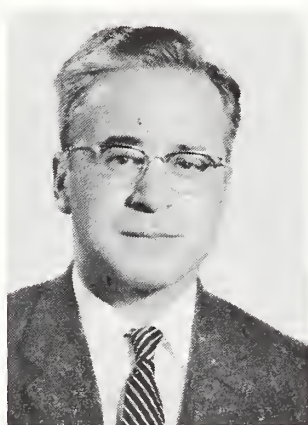
Maine Society of Obstetrics and Gynecology  
PHILIP H. McCrum, M.D., Portland, presiding

### The Borderline Pelvis

CHRISTOPHER J. DUNCAN, M.D., Brookline, Massachusetts



Dr. Duncan



Dr. Fasanella

Maine Chapter of the American Academy of Pediatrics

HENRY C. THACHER, M.D., Auburn, presiding

### Subject to be announced

SAUL KRUGMAN, M.D., New York, New York

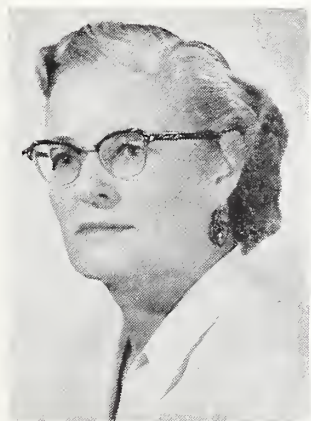
M.M.A. Eye Section

CAROL SCHWARTZ, M.D., Portland, presiding

### Lacrimal Surgery

R. M. FASANELLA, M.D., New Haven, Connecticut

Film: Cataract Surgery



Dr. Rodger



Dr. Wroblewski

Maine Society of Clinical Hypnosis

SIMON C. BEAUDET, M.D., Lewiston, presiding

### Hypnosis in the Conquest of Inner Space

BERTHA RODGER, M.D., Ridgewood, New Jersey

Maine Society of Pathologists

ROBERT D. WAKEFIELD, M.D., Lewiston, presiding

### Enzymology

FELIX WROBLEWSKI, M.D., New York, New York

Maine Thoracic Society

DAVID DAVIDSON, M.D., Portland, presiding

### Solitary Lung Lesion

EARLE W. WILKINS, JR., M.D., Boston, Massachusetts



Dr. Wilkins

**Tuesday, June 19**

10:00 A.M. Maine Medico-Legal Society

Business Meeting

Election of Officers — Committee Reports —  
Proposed changes in Constitution and By-Laws

2:00 P.M. to 4:00 P.M.

Maine Society of Anesthesiologists

ALDEN W. SQUIRES, M.D., Togus, presiding

### Operating Room Safety

GEORGE J. THOMAS, M.D., Pittsburgh, Pennsylvania

## Maine Society of Internal Medicine

PAUL H. PFEIFFER, M.D., Waterville, presiding

**King Anderson Bill — Prelude to the Future**

OSLER L. PETERSON, M.D., Boston, Massachusetts



Dr. Peterson

## Maine Radiological Society

Annual Business Meeting

ALBERT A. POULIN, M.D., Waterville, presiding

**Luncheon Meetings****Monday, June 18**

Amy W. Pinkham Fund Committee

**Tuesday, June 19**

Maine Radiological Society

**SPECIAL NOTICES****Election of President-Elect**

The election of a President-Elect will take place at the General Assembly, June 18 at 4:00 P.M.

**Election of Councilors**

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 17 at 3:30 P.M.

Third District — Knox and Lincoln-Sagadahoc Counties

Fourth District — Kennebec, Somerset and Waldo Counties

In accordance with the By-Laws, "Nominations for members of the Council for any District where there is a vacancy shall be made by a caucus of the members of the House of Delegates in that District. Each candidate for Councilor must be a resident of the district for which he is nominated."

**Council Meetings**

The Council will meet on Saturday, June 16 at 3:30 P.M. and daily throughout the session at a time and place to be announced.

**Golf Tournament**

DANIEL R. SHIELDS, M.D., Chairman

**HONORARY PINS**

Presentation of the Association's Honorary Pins will be made by James A. MacDougall, M.D., President, at the Annual Banquet, Monday evening, June 18 at 6:30 P.M.

**FIFTY-YEAR PINS**

Fifty-Year Lapel Pins will be presented to the following members who were graduated from Medical School in 1912:

**Androscoggin County**

Horace L. Gauvreau, M.D., Lewiston  
University of Vermont Medical School

Harold S. Pratt, M.D., Livermore Falls  
Bowdoin Medical School

**Kennebec County**

J. Ramser Crawford, M.D., Augusta  
Memphis Hospital Medical College

**Lincoln-Sagadahoc County**

Arthur U. Desjardins, M.D., South Bristol  
University of Pennsylvania

**Oxford County**

Nathaniel Mills, M.D., Harrison  
Johns Hopkins University School of Medicine

Harold W. Stanwood, M.D., Dixfield  
Bowdoin Medical School

**Penobscot County**

George B. Weatherbee, M.D., Hampden Highlands  
Tufts University School of Medicine

**Waldo County**

Foster C. Small, M.D., Belfast  
University of Vermont Medical School

### FIFTY-FIVE-YEAR PINS

Fifty-Five-Year Pins will be presented to the following members who received Fifty-Year Medals in 1957:

#### Kennebec County

Charles H. Newcomb, M.D., Clinton  
Maurice A. Priest, M.D., Deland, Florida

#### York County

Joseph R. Larochelle, M.D., Biddeford

### SIXTY-YEAR PIN

#### Franklin County

George L. Pratt, M.D., Farmington

### Visiting Delegates

Connecticut State Medical Society  
JACK GURWITZ, M.D., Hartford  
SYDNEY LURIA, M.D., Bridgeport

Massachusetts Medical Society  
ALLEN S. JOHNSON, M.D., Longmeadow

New York Medical Society

SAMUEL Z. FREEDMAN, M.D., New York

Rhode Island Medical Society

HANNIBAL HAMLIN, M.D., Providence

Vermont State Medical Society

W. HERBERT JOHNSTON, M.D., Montpelier

### Delegates to Out-of-State Meetings

Connecticut State Medical Society

PETER B. AUCOIN, M.D., Rumford

Massachusetts Medical Society

ALBERT P. ROYAL, JR., M.D., Rumford

New York Medical Society

JAMES A. MACDOUGALL, M.D., Rumford

Rhode Island Medical Society

GEORGE C. HOWARD, M.D., Guilford

Vermont and New Hampshire Medical Societies

JAMES A. MACDOUGALL, M.D., Rumford

GEORGE E. SULLIVAN, M.D., Fairfield

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### Program for the Ladies

Monday, June 18, 9:30-11:00 A.M.

Coffee — Penobscot Room

Tuesday, June 19, 9:30-11:00 A.M.

Coffee — Penobscot Room

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Evening Programs — See Maine Medical  
Association Official Program

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### Scientific and Educational Exhibits

A.M.A. Department of Foods and Nutrition

Associated Hospital Service of Maine

Beacon Investing Corporation, Rutland, Vermont

Division of Cancer Control of the State of Maine  
Department of Health and Welfare

Maine Cancer Society

Unproved Cancer Therapy — A Continuing  
Challenge

Maine Chapter, American Academy of General  
Practice

Maine Heart Association

Maine League for Nursing

Maine Medical Center

Maine Society of Clinical Hypnosis

Maine Society of Internal Medicine

Maine Society of Obstetrics and Gynecology

Maine State Nurses' Association

Maine Trauma Committee

## County Delegates

### FIRST DISTRICT

#### Cumberland County Medical Society

*Delegates* — Albert Aranson, M.D., 39 Deering St., Portland — Secretary

(2 years)

Charles R. Glassmire, M.D., 58 Deering St., Portland

David K. Lovely, M.D., 46 Deering St., Portland

Robert H. Pawle, M.D., 8 Walcott Ave., Falmouth

Robinson L. Bidwell, M.D., 31 Bramhall St., Portland (1 year)

Philip S. Fogg, Jr., M.D., 173 Pleasant Ave., Portland

Morrill Shapiro, M.D., 29 Deering St., Portland

David S. Wyman, M.D., 47 Deering St., Portland

Benjamin Zolov, M.D., 296 Congress St., Portland

Emerson H. Drake, M.D., 18 Bramhall St., Portland

#### *Alternates*

(2 years)

George O. Chase, M.D., 144 State St., Portland

John F. Gibbons, M.D., 22 Bramhall St., Portland

Donald P. Cole, M.D., 45 Deering St., Portland

Ronald A. Bettie, M.D., 32 Federal St., Brunswick (1 year)

Donald E. Allen, M.D., Sebago Lake

Eben T. Bennett, M.D., 49 Deering St., Portland

Louis G. Bove, M.D., 12 Deering St., Portland

Maurice Van Lonkhuyzen, M.D., 31 Bramhall St., Portland

Howard P. Sawyer, Jr., M.D., 22 Bramhall St., Portland

#### York County Medical Society

*Delegates* — Charles W. Kinghorn, M.D., 4 Wentworth St., Kittery — Secretary

Robert F. Ficker, M.D., Maine St., Kennebunkport

Roger J. P. Robert, M.D., 331 Main St., Saco

Carl E. Richards, M.D., 34 Winter St., Sanford

#### *Alternates*

Kenneth E. Leigh, M.D., Brixham Rd., York

Stephen A. Cobb, M.D., 34 Winter St., Sanford

Melvin Bacon, M.D., 122 Main St., Sanford

### SECOND DISTRICT

#### Androscoggin County Medical Association

*Delegates* — Donald L. Anderson, M.D., 369 Main St., Lewiston — Secretary

Paul J. B. Fortier, M.D., 111 Webster St., Lewiston

(1 year)

Waldo A. Clapp, M.D., 215 College St., Lewiston

(2 years)

Harvey J. Proulx, M.D., 92 Pine St., Lewiston (2 years)

Louis N. Fishman, M.D., 327 Main St., Lewiston

(3 years)

#### *Alternates*

Frederick B. Lidstone, M.D., 117 Goff St., Auburn (1 year)

Wilfrid A. Cloutier, M.D., 210 Sabattus St., Lewiston

(2 years)

Edward L. Reeves, M.D., 179 Sabattus St., Lewiston

(2 years)

Charles A. Hannigan, M.D., 85 Goff St., Auburn

(3 years)

#### Franklin County Medical Society

*Delegates* — Philip B. Chase, M.D., 36 Main St., Farmington — Secretary

Wallace H. Duffy, M.D., 100 Main St., Farmington

#### *Alternate*

Paul E. Floyd, M.D., 2 Middle St., Farmington

#### Oxford County Medical Society

*Delegates* — Albert P. Royal, Jr., M.D., 82 Maine Ave., Rumford — Secretary

John A. Greene, M.D., 96 Congress St., Rumford

(1 year)

Joelle C. Hiebert, Jr., M.D., Box 148, Norway (2 years)

#### *Alternates*

H. Richard Bean, M.D., 171 Main St., Norway (1 year)

Dexter E. Elsemore, M.D., 11 Main St., Dixfield

(2 years)

### THIRD DISTRICT

#### Knox County Medical Society

*Delegates* — Mustafa V. Onat, M.D., St. George — Secretary

Albert L. Hunter, M.D., Knox County General Hospital, Rockland

Merrill J. King, Jr., M.D., 22 White St., Rockland

#### *Alternate*

Johan Brouwer, M.D., 56 Talbot Ave., Rockland

#### Lincoln-Sagadahoc County Medical Society

*Delegates* — George W. Bostwick, M.D., Newcastle — Secretary

Ralph C. Powell, M.D., Damariscotta

John F. Andrews, M.D., 20 West St., Boothbay Harbor

#### *Alternates*

Mary J. Tracy, M.D., Bristol Rd., Damariscotta

Miriam C. Doble, M.D., 990 Washington St., Bath

### FOURTH DISTRICT

#### Kennebec County Medical Association

*Delegates* — Earle M. Davis, M.D., 2 School St., Waterville — Secretary

Anthony E. Lepore, M.D., 72 Church St., Gardiner

Francis J. O'Connor, M.D., 4 Woodlawn St., Augusta

Paul H. Pfeiffer, M.D., 14 Gilman St., Waterville

Joseph A. Marshall, M.D., 177 Main St., Waterville

Philip Dachslager, M.D., 21 Western Ave., Augusta

#### *Alternates*

John D. Denison, M.D., 105 Brunswick Ave., Gardiner

Lane Giddings, M.D., 6 E. Chestnut St., Augusta

Kenneth W. Sewall, M.D., 2 School St., Waterville

Sampson Fisher, M.D., 173 Main St., Waterville

Napoleon J. Gingras, M.D., 6 E. Chestnut St., Augusta

#### Somerset County Medical Society

*Delegates* — Harland G. Turner, M.D., R.F.D. #2, Norridgewock — Secretary

George E. Sullivan, M.D., R.F.D. #1, Fairfield

#### *Alternate*

Howard L. Reed, M.D., 68 Water St., Skowhegan

#### Waldo County Medical Society

*Delegates* — Seth H. Read, M.D., 15 Church St., Belfast — Secretary

Carl H. Stevens, M.D., 18 Franklin St., Belfast

#### *Alternate*

Norman E. Cobb, M.D., 132 Main St., Belfast

**FIFTH DISTRICT****Hancock County Medical Society**

*Delegates* — Russell G. Williamson, M.D., Blue Hill Memorial Hospital, Blue Hill — Secretary  
Llewellyn W. Cooper, M.D., 194 Main St., Bar Harbor  
Elizabeth E. Williamson, M.D., Blue Hill

*Alternates*

Arthur M. Joost, Jr., M.D., P. O. Box B, Bucksport  
Philip L. Gray, M.D., Blue Hill

**Washington County Medical Society**

*Delegates* — Karl V. Larson, M.D., East Machias — Secretary

John W. McAllister, M.D., Lubec

*Alternate*

Hazen C. Mitchell, M.D., Calais

**SIXTH DISTRICT****Aroostook County Medical Society**

*Delegates* — Clyde I. Swett, M.D., 18 Sherman St., Island Falls — Secretary

Frederick J. Gregory, M.D., 16 High St., Caribou

Raymond G. Giberson, M.D., 156A Academy St., Presque Isle

Arthur K. Carton, M.D., Market Square, Houlton

*Alternates*

Samuel Rideout, M.D., 3 Green St., Fort Fairfield  
Thomas V. Brennan, M.D., 99 Hardy St., Presque Isle  
H. Douglas Collins, M.D., Caribou Clinic, Caribou

**Penobscot County Medical Association**

*Delegates* — Frederick C. Emery, M.D., 242 Cedar St., Bangor — Secretary

Charles D. McEvoy, Jr., M.D., 316 State St., Bangor

John J. Pearson, M.D., 100 So. Main St., Old Town

Irvin E. Hamlin, M.D., Main St., East Millinocket

Arthur N. Lieberman, M.D., 180 Broadway, Bangor

George W. Wood, III, M.D., 156 No. Main St., Brewer

*Alternates*

Nelson P. Blackburn, M.D., 489 State St., Bangor

Leonard G. Miragliuolo, M.D., 10 Maple St., Bangor

William M. Shubert, M.D., 317 State St., Bangor

Philip B. Thomas, M.D., 205 French St., Bangor

William W. Purinton, M.D., 15 Ohio St., Bangor

**Piscataquis County Medical Society**

*Delegates* — Isaac Nelson, M.D., Box 336, Greenville — Secretary

Linus J. Stitham, M.D., 50 Main St., Dover-Foxcroft

*Alternate*

Charles H. Lightbody, M.D., No. Main St., Guilford

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## Technical Exhibits

**Abbott Laboratories, North Chicago, Illinois**

Representatives: Mr. A. J. Mack, Mr. A. Tancredi,  
Mr. W. A. Towne, Mr. D. K. Eastman

**The Alkalol Company, Taunton, Mass.**

Representative: Mr. E. W. LeClair

**Ayerst Laboratories, 245 Paterson Ave., Little Falls, New Jersey**

Representatives: Mr. Edward McMahon, Mr. Stanley Hewson

**The Baker Laboratories, Inc., 3940 Euclid Ave., Cleveland 15, Ohio**

Representative: Mr. Henry C. Nicoll

**Bicknell Photo Service, 517 Congress St., P. O. Box 2011, Portland, Maine**

Representative: Mr. Fred E. Wormell, Jr.

**Elmer N. Blackwell, Surgical Appliance Specialist, 565 Congress St., Room 207, Portland, Maine**

Representatives: Mr. Elmer N. Blackwell, Mr. Oakley R. Sanborn

**The Borden Company, 350 Madison Ave., New York 17, N. Y.**

Representatives: Mr. Joseph R. Galvin, Mr. George Wagner

**Brewer & Company, Inc., 67 Union St., Worcester 8, Mass.**

Representatives: Mr. Sidney L. Segel, Mr. Walter Spaulding

**Buffington's Inc., Worcester 8, Mass.**

Representative: Mr. C. W. Rich

**Burroughs Wellcome & Co. (U.S.A.) Inc., 1 Scarsdale Rd., Tuckahoe, New York**

Representatives: Mr. L. C. Gee, Mr. William Murley, Mr. Richard Parke

**Carnation Company, 5045 Wilshire Blvd., Los Angeles 36, California**

Representatives: Mr. Russell B. Mundi, Mr. John J. Burke, Jr., Mr. William L. Galatas, Mr. Arthur K. Groppe, Mr. Robert L. Garofano

**Ciba Pharmaceutical Products Inc., Summit, New Jersey**

Representatives: Mr. John H. Angis, Mr. John Sullivan

**The Coca-Cola Company, P. O. Drawer 1734, Atlanta 1, Georgia****Eaton Laboratories, Norwich, New York**

Representatives: Mr. A. Snyder, Mr. D. M. Woodward

**Endo Laboratories Inc., 84-40 101st St., Richmond Hill 18, New York**

Representatives: Mr. David D. Green, Mr. George Williams, Mr. Robert Prescott

**Geo. C. Frye Company, 116 Free St., Portland, Maine**

Representatives: Mr. Sidney F. Cheney, Mr. Irving F. Beers, Mr. Millard C. Webber, Mr. Hubert A. Honan, Mr. Milton S. Kimball, Mr. John F. Kimball, Mr. Arthur R. Wickham

**Geigy Pharmaceuticals, P. O. Box 430, Yonkers, New York**

**Hayden, Stone & Co., Inc., Casco Bank Building, Portland, Maine**

Representatives: Mr. Lawrence M. Burke, Mr. Vere B. Crockett

**Holland-Rantos Company, Inc., 145 Hudson St., New York 13, N. Y.**

Representative: Mr. Milton Hart

**Lederle Laboratories, Pearl River, New York**

Representatives: Mr. R. Maffei, Mr. Jean Boucher, Mr. James E. Crosby, Mr. Gregory Pooler

**E. F. Mahady Company, 225 Monsignor O'Brien Highway, Cambridge 41, Mass.**

Representatives: Mr. Robert Blair, Mr. Charles Perkins

**Maine Surgical Supply Co., 233 Vaughan St., Portland, Maine**

Representatives: Mr. Philip Dana, Jr., Mr. John H. Lacy, Mr. George Munroe, Mr. Robert Axelsson, Mr. Lawrence Gardiner, Mr. Lewis Olore

**The S. E. Massengill Company, Inc., 717 Fifth Ave., New York 22, N. Y.**

Representatives: Mr. R. F. Blais, Mr. L. C. Miller

**McNeil Laboratories, Inc., Camp Hill Road, Fort Washington, Pa.**

Representatives: Mr. George A. Stevens, Mr. A. W. Hunt, Mr. G. Behrakis, Mr. R. Bender, Mr. J. R. Marshall, Mr. C. C. Kantar

**Mead Johnson Laboratories, Evansville 21, Indiana**

Representative: Mr. Kendall Dow

**The Wm. S. Merrell Company, Cincinnati 15, Ohio**

Representatives: Mr. Joseph F. Crozier, Mr. James R. MacIsaac

**The National Drug Company, 4663 Stenton Ave., Philadelphia 44, Pa.**

Representative: Mr. William P. Dunbar

**Parke, Davis & Company, Detroit 32, Michigan**

Representatives: Mr. Walter Nikitin, Mr. Gene Giroux

**Pfizer Laboratories, 235 East 42nd St., New York 17, N. Y.**

Representatives: Mr. Wallace Houston, Mr. L. Robinson, Mr. B. Garcelon

**Plough Laboratories, Inc., 3022 Jackson Ave., Memphis, Tennessee**

Representative: Mr. Fred Casey

**Riker Laboratories, Inc., Northridge, California**

Representatives: Mr. Louis Celantano, Mr. John Cella

**A. H. Robins Company, Inc., 1407 Cummings Drive, Richmond 20, Virginia**

Representatives: Mr. Charles Kokernak, Mr. Stephen Owen, Mr. Frederick O'Brien

**Roche Laboratories, Nutley 10, New Jersey**

Representatives: Mr. Karl Norris, Mr. Paul Dickey

**J. B. Roerig and Company, 235 East 42nd St., New York 17, N. Y.**

Representative: Mr. Clarence J. Johnson

**William H. Rorer, Inc., 500 Virginia Drive, Fort Washington, Pa.**

Representatives: Mr. E. T. Croke, Mr. J. Beward

**Ross Laboratories, Columbus 16, Ohio**

Representatives: Mr. Harold Hutchinson, Mr. Richard Kaufman

**Sandoz Pharmaceuticals, Hanover, New Jersey**

Representative: Mr. Frank Powers

**W. B. Saunders Company, West Washington Square, Philadelphia 5, Pa.**

Representative: Mr. Joseph Juneman

**Schering Corporation, Bloomfield, New Jersey**

Representatives: Mr. Jack Arlaud, Mr. Floyd Selby

**G. D. Searle & Company, P. O. Box 5110, Chicago 80, Illinois**

Representatives: Mr. James H. Muncaster, Mr. Alfred L. Grimes, Mr. John J. Pash

**Smith, Miller & Patch, Inc., 902 Broadway, New York 10, N. Y.**

Representatives: Mr. Paul Woodward, Mr. Kenneth Mullen

**E. R. Squibb & Sons, 745 Fifth Ave., New York 22, N. Y.**

Representatives: Mr. J. L. Jameson, Mr. Bill Gray

**Surgeons' and Physicians' Supply Co., 961 Commonwealth Ave., Boston 15, Mass.**

Representatives: Mr. Lester E. Clough, Mr. John R. Stutz

**U. S. Vitamin & Pharmaceutical Corporation, 800 Second Ave., New York 17, N. Y.**

Representatives: Mr. William G. Moran, Jr., Mr. John R. Winfield

**Warner-Chilcott Laboratories, Morris Plains, New Jersey**

Representative: Mr. William H. Comyns

**The Warren-Teed Products Company, 582 West Goodale St., Columbus 15, Ohio**

Representative: Mr. Robert W. Roffler

## Committee Reports — 1961-1962

### Committee On Recruitment, Aid And Placement

The Committee has continued to concern itself with the Medical Education Foundation. This coming year should see some major additions to the Maine Medical Education Foundation from the general public and from philanthropic organizations.

A meeting of the Committee in April, 1962 was attended by Doctors Hanley, Barrett, Capron and Pfeiffer and at this meeting a suggestion was made by Dr. Capron that the funds in the Medical Education Foundation could serve a greater number of applicants if the funds were used to guarantee bank loans and to pay the interest on the loans. This appears to have a great deal of merit and Dr. Capron is pursuing this idea with one of the Banks in Portland as well as the Maine Bankers' Association and the results of his investigation will be presented formally at the House of Delegates meeting in June.

The Committee has continued to be active in attempts to place physicians in the State of Maine. Letters have been written to physicians who have expressed an interest in practicing in this State. The Cumberland County Medical Society has been particularly active in going to the secondary schools and presenting the point of view of medicine as a career and encouraging the development of premedical clubs in schools. This type of activity is also being done in the Bangor area.

It is hoped that each physician will contact the local school system and make every effort to encourage students who are interested in medicine or science as a way of life.

PAUL H. PFEIFFER, M.D., *Chairman*

### Public Relations Committee

At a recent meeting in Brunswick, this Committee reviewed the entire public relations situation at the local, state and national levels. We feel that the present very important problems confronting the medical profession at all levels have never been more critical.

We feel that these problems are deserving of very careful thought and planning and that it is absolutely essential to maintain the highest quality possible in public relations. We feel that our public relations program should be carried out by professionals in this field on a full time basis.

We realize that this would be a costly venture, but we feel that at this critical time, it is a matter of necessity and should be given very careful consideration by the Maine Medical Association.

DONALD F. MARSHALL, M.D., *Chairman*

### Rural Health Committee

The Rural Health Committee met four times during 1961-1962: in June at Rockland with Dr. Jean A. Curran and Mr. George Nilson; in October at Augusta; in December at Augusta with all but one Rural Health Committee member, plus Dr. Ella Langer; in April again at Augusta and with Dr. Langer. As Chairman, I attended the New England Rural Health Conference in Concord, and the Annual Meeting of the Council of the New England State Medical Societies.

At these meetings we discussed many subjects, including immunization clinics, child health conferences, rural doctor recruitment, continuation education for medical doctors and osteopaths in rural areas. Our discussions did not reach any conclusions, so that the Rural Health Committee does not have any recommendations for the House of Delegates, although I feel we all profited by the exchange of ideas.

I would like to thank each of the committee members for excellent cooperation during the past year, and I anticipate that the Rural Health Committee will continue to increase its activities.

ROBERT H. PAWLE, M.D., *Chairman*

### Committee On Conservation Of Vision

Discussion has been carried out by the Committee this year on the advisability of the Maine Medical Association distributing to its members, and to individuals responsible for health programs in the State, information put out by the National Foundation for Medical Eye Care.

Over the years a group of non-medical practitioners, namely optometrists, have promoted the impression to the public that they are the custodians of eye care. They have been aggressive in advising school boards on vision screening tests and eye examination for students. They have entered the medical field by diagnosing eye diseases. In their contact lens work they have entered another field of medical concern — one whose essentiality is extremely limited — but nevertheless one in which the medical implications are very real. In some states ophthalmologists have been harassed by optometrists' groups attempting to obtain legislation that would make it illegal for ophthalmologists to refract patients.

Therefore, for the benefit of the public in the conservation of vision, it is advisable that all M.D.'s keep informed on these subjects in order to give advice whenever there is occasion to advise and to support more effectively their colleagues, the ophthalmologists, as an integral part of the medical team in the State. The National Foundation of Medical Eye Care is an authoritative source of such information. This organization was founded by ophthalmologists for the purpose of research in eye care for the public and for the dissemination of information about it.

No decision has been made by the Committee for requesting the Maine Medical Association to provide such information service to its members and to individuals responsible for public health programs (including school superintendents in the matter of eye examinations in schools), but this idea will be further explored.

DEXTER J. CLOUGH, 2nd, M.D., *Chairman*

### Committee On Mental Health

As in preceding years, this Committee has been inactive for lack of direction, planning and realistic goals. This, however, is not a unique characteristic of this Committee or State, since there is much activity at the A.M.A. level to stimulate State, County and Community effort in the Mental Health and Illness program. To implement this, the First American Medical Association National Congress on Mental Health was held in September 1961 when "175 experts" gathered to

"formulate a platform in which physicians of the United States may build in the ensuing years."

As an outgrowth of this, the 8th Annual Conference, with theme of "Implementation of the A.M.A.'s Program on Mental Illness and Health" was held in February, 1962. The Chairman of this Committee did not attend, but the proceedings are to be received shortly.

At the State of Maine level, however, there has been much continued and increased effort in this direction. Most notable and laudable, is the introduction of the "New Mental Hospital Admissions Law" which has served to facilitate hospitalization of a patient with emphasis on the medical rather than the legal aspects of commitment. The voluntary admissions have thereby increased markedly. Further, a rather significant substitution in the Law relating to criminal responsibility from the M'Naghten Rule to the Durham Rule, which is more consistent with contemporary psychiatric thinking.

Dr. William Schumacher has been active in his capacity as Director of the Bureau of Mental Health as a stimulus, among many other things, for the expansion of Community Health Services. There are "presently nine Community activities in the Mental Health area and nine services for the trainable retarded children under our community health subsidy law." As of July, 1962, the Division of Mental Health of the Department of Health and Welfare will be co-ordinated under the Bureau of Mental Health of the Department of Mental Health and Corrections, a coordination of Mental Health Services. This Department sponsored a two-day seminar presented by the Social Psychiatry Department of Cornell University, under the leadership of Dr. Alexander Leighton. Dr. O. Spurgeon English has agreed to present a similar seminar this year.

Dr. Harold A. Pooler, Superintendent of the Bangor State Hospital, describes continued progressive activity in this hospital: in summary, the geriatric pavillion was completed with the new addition, housing an additional 187 patients; the services of two alcoholics have been expanded by the ready admission and a continued In Patient AA Group; enlarged Out Patient services; the addition of an educator to the Staff to conduct an educational and rehabilitation program. Voluntary admissions are running as high as "forty-five percent" at this hospital.

Dr. Francis H. Sleeper, Superintendent of the Augusta State Hospital, has reason to be proud of the "Unconditional Accreditation" for three years by the Joint Commission. Increase in voluntary admissions is noted here, also, and he states that the admission rate this year will be the highest in the history of the institution.

Although no information has been received specifically from Dr. Bowman at the Pineland Hospital, it is known that they have expanded their services by the opening of the Children's Psychiatric Hospital this past year.

There has been no meeting of the Committee this year.

GUY N. TURCOTTE, M.D., *Chairman*

### Diabetes Committee

#### Report of Diabetes Education and Detection Drive in State of Maine, 1961

It was a considered privilege and pleasure to serve as the chairman of the Diabetes Committee of the Maine Medical Association of 1961. I must say that the cooperation given me by the physicians, Bureau of Health and Welfare, under the direction of Miss Ruth Clough, Health Consultant, the Maine Pharmaceutical Association, Hospitals, Colleges, Industry, Service Installations, Daniel F. Hanley, M.D., Executive Director of the Maine Medical Association, Public and Parochial Schools, etc., was superb. Their cooperation was un-

surpassed. The Diabetes Detection and Education Program could not have achieved the proportions that it did without their help and assistance.

A chairman was chosen for each county in the State and the program was coordinated with the State Bureau of Health and Welfare as a combined effort. Radio, Television, Pamphlets, Newspapers, Diabetes Fairs, Posters, Movies, and personal contact were media used.

The following chart indicates the type program used in this endeavor; the number tested for Glycosuria and the number of positives found in the State of Maine:

Chart I — Results of 1961 Diabetes Detection Drive in the State of Maine:

Type of Program	Number of persons Tested for sugar	Number of Positive Tests for Glycosuria
Industry	1,355	82
Hospitals	36,603	688
Physicians	12,666	319
Chairmen	1,784	28
Government Installations (Dow & Brunswick Airbases)	19,930	397
Colleges	2,127	
Bureau of Health & Welfare	8,268	106
<b>TOTAL</b>	<b>82,733</b>	<b>1,620</b>
York County	19,442	115
	<b>102,175</b>	<b>1,735</b>

There were 102,175 Specimens checked for Glycosuria in the State and 1,735 positives found.

As the York County Program was somewhat more detailed, the total figures were added to the total of the rest of the State as indicated in Chart I; and a breakdown of the figures for York County given in the following chart:

Chart II — Results of 1961 Diabetes Detection Drive in York County:

Type of Program	Number of Persons tested for sugar	Number of Positive tests for glycosuria	Known Diabetics	Unknown or newly found Diabetics
Private Physicians	1,069	47	32	13
Schools				
(Parochial & Public)	5,704	9		2
Drug Stores	306	4		4
Industries	10,726	13		13
Diabetes Fair (Sanford)	325	25	19	6
Hospitals	648	8	3	
Detection Centers	476	5	2	3
Town Employees (Sanford)	36			
College & Faculty (Notre Dame Institute)	127	2		
Town Farm (Sanford)	25	2	2	
	<b>19,442</b>	<b>115</b>	<b>58</b>	<b>41</b>

In York County there were 19,442 specimens checked for glycosuria and 115 positives found. Of the totals checked, there were 58 known diabetics and 41 unknown or newly discovered diabetics. The Diabetes Detection and Education Program was carried on in part as a year-round program. However, for the most part, the remainder was the result of a considered effort during Diabetes Week, November, 1961.

In closing, may I again reiterate my thanks to all the participants in the Diabetes program. "As goes Maine, so goes the Nation, Diabetically speaking."

MELVIN BACON, M.D., *Chairman*

### Committee For Maternal And Child Welfare

A committee meeting was held in Augusta, October 20, 1961 attended by Doctors William Shubert, Ella Langer and Alice A. S. Whittier. The hospital obstetric forms available from the Department of Medical Service, American Medical Association, were examined and the possibility of introducing these into the larger hospitals was discussed. It was thought that it would be difficult to use the detailed Obstetricians Newborn Record in most of the hospitals. It seemed as though the use of the Newborn Infant Record was more possible. This has not been followed up as yet.

Sending an Information Statement on Neonatal Jaundice, prepared by the Committee on Fetus and Newborn of the American Academy of Pediatrics, to the nurseries of the hospitals in the State is being considered and these will be mailed shortly provided the members of the Committee approve.

Alice A. S. Whittier, M.D., *Chairman*

### Arthritis Committee

The activities of the Pine Tree Arthritis and Rheumatism Foundation have supported several postgraduate education seminars throughout the state during the past year. Attempts have been made to organize other local Rheumatism Societies without success as yet.

Mobile Physical Therapy visit in the greater Portland area functioned well until the therapist left to join the Armed Forces. Services of a new therapist are being sought.

The facilities for rehabilitation in Bath and Waterville are being utilized for arthritic patients.

Philip P. Thompson, Jr., M.D., *Chairman*

### Amy W. Pinkham Fund Committee

This year instead of merely listing the grants made by the Amy W. Pinkham Fund, I think it would be nice to make a report of who was Amy Pinkham.

This information has been obtained from Mr. Frank E. Mott of West Roxbury, Massachusetts, who for many years was milk commissioner of Boston. Frank Mott's wife was Amy Pinkham's sister.

The following is copied from a letter received from Mr. Frank E. Mott, 75 Vermont Street, West Roxbury 32, Massachusetts, dated April 11, 1962;—

"Amy W. Pinkham was the daughter of Sarah Turner of Millbridge, Maine, who married Captain Elijah Jackson Pinkham of South Harpswell, Maine,—later of Portland, Maine.

"Amy died in November 1941. Her mother, Sarah Turner, before her marriage had taught school in rural districts in Eastern Maine and by direct contact knew about undernourished or tuberculous children in that area, and she planned with Amy that Amy might well bequeath money to aid such children if circumstances should develop so that it would appear wise to make a Will.

"Amy was a semi-invalid who trained at Castine Normal School and was certified by the State of Maine to teach in rural districts. Because of poor health she never taught school.

"Amy's sister, Bertha P. Mott, of Boston, Massachusetts, died July 11, 1941, and so Amy was the sole heir of the Estate of Sarah Turner Pinkham.

"Amy asked her brother-in-law, Frank E. Mott, of Boston to help her to make her Will. He drew up the Will and

so far as relates to the above '*Undernourished or Tuberculous Children of Maine*' he suggested that possibly the best way to accomplish her desires would be to give the money to '*The Maine Public Health Association*.'

"Frank E. Mott was Executor of the Will which reads: 'I give and bequeath to the Maine Public Health Association, Inc., twenty thousand dollars to the use of the *Undernourished or Tuberculous Children of Maine*. Preferably those Children from Rural Districts.'

"The Maine Public Health Association, Inc., by vote of its board of directors, under seal, '*Accepted the Bequest in Trust*' and in writing pledged itself to Judge Chaplin of the Probate Court of Cumberland County 'to engage one of the best Trust Depositories to handle the twenty thousand dollars and to disburse the income *only* in compliance with the Will.'

"It was further decreed that executor's selection of Maine Public Health Association, Inc., does receive consent of Court on condition said Maine Public Health Association, Inc., is willing and will agree to accept said bequest for the purposes named in Will, the principal of the bequest to be kept safely invested and the income to be used for the purposes named in said Will after consultation by said Maine Public Health Association, Inc., with a committee of the Maine Medical Association appointed for said purpose."

The Maine Public Health Association later became the Maine Tuberculosis & Health Association which now holds this bequest.

Mr. Mott consulted Edwin T. Wyman, M.D., a pediatrician of Boston who was born in Sebec, Maine, and who still has a summer home on Sebec Lake, as to how the purposes of this Will could best be fulfilled.

It was their opinion that this could best be accomplished by promoting the use of pasteurized milk in school lunches. At that time pasteurized milk was not generally used as it is today. I remember that Dr. Wyman, when he was talking to Mr. Mott regarding this Will, said he was ashamed of his favorite county—Piscataquis—because he had not been able that summer to buy pasteurized milk in either Dover-Foxcroft, or Milo, the two largest towns in the County.

All grants to schools were made subject to the provision that only pasteurized milk would be used in the school lunches, thus promoting the use of pasteurized milk in the State of Maine.

It is hoped that Frank Mott, Dr. Wyman and Mr. Wells of the Maine TB & Health Association will meet with the committee from the Maine Medical Association on June 18 of this year at The Samoset, and at that time, any member of the Association who has ideas as to how this money could be better expended will have an opportunity to present such ideas to the Committee, Dr. Wyman, Frank Mott and Mr. Wells.

Since May of 1951, grants totaling \$7,912.16 have been made by this Fund to schools in rural areas in the State of Maine.

Norman H. Nickerson, M.D., *Chairman*

### Committee On Clinical Hypnosis

At the 1961 annual session of the House of Delegates, Maine Medical Association, a resolution was adopted relating to the practice of medical hypnosis. This resolution provided for the appointment of this committee to study and prepare a proposed Bill for presentation to the next Maine State Legislature.

This committee has met and corresponded on several occasions during the past year and certain studies were made, including documents and legislative acts already adopted by several other states and municipalities. From this study, the

committee reached certain conclusions before drafting the proposed Bill presented in this report.

It was also learned that the outstanding leaders in the field of medical and experimental hypnosis in this country, the American Society of Clinical Hypnosis, the Society for Clinical and Experimental Hypnosis, the American Medical Association, the British Medical Association, the Maine Society of Clinical Hypnosis, and others, have accepted hypnosis as an ethical and effective procedure in the fields of medicine, dentistry, and psychology. All of the above agencies emphatically condemn the use of hypnosis by layman and others for purposes of entertainment.

This committee feels that such unprofessional use of hypnosis can be harmful to the citizens of Maine and that it is a major responsibility of this Association to protect the health and welfare of the people in this respect. Accordingly, this committee submits the following proposed Legislative Bill for adoption by the House of Delegates of the Maine Medical Association at its annual session, held in Rockland, Maine, in June 1962:

AN ACT TO PROTECT THE PUBLIC FROM THE USE OF  
HYPNOSIS BY UNQUALIFIED PERSONS:

Sec. 1: If any person shall hypnotize or attempt to hypnotize any person, he shall be guilty of a misdemeanor. But this section shall not apply to hypnotism performed by legally accredited doctors of medicine, dentistry, and

psychology; provided, however, that such qualification is in accord with proper medical, dental and psychological professional requirements. Psychologists shall utilize hypnosis for therapeutic purposes only in accordance with existing laws.

Sec. 2: Misdemeanor, for which no punishment or no maximum punishment is prescribed by statute, shall be punished by fine not exceeding five hundred dollars or confinement in jail not exceeding twelve months, or both, in the discretion of the jury or judge.

Sec. 3: The Maine Board of Registration of Medicine for licensure and regulation of medical doctors, and the similar licensing bodies for osteopathic physicians, dentists, and accredited psychologists, shall administer and enforce this Act and may designate the Office of the Attorney General, which may appoint any legally qualified medical practitioner for the purpose of making any investigation or inquiry necessary therefor.

Sec. 4: Every prosecution under this Act shall be commenced within one year from the date of alleged offense.

Sec. 5: The restrictions of Section 1 do not apply to any bona fide student registered in a course leading to qualification in one of the professions named in the Section practicing hypnosis for the purpose of study under the instruction and supervision of a legally accredited doctor of medicine, dentist, or psychologist.

CLYDE I. SWETT, M.D., *Chairman*

## In Memoriam

### *Androscoggin County*

Everett C. Higgins, M.D.

Lewiston

### *Aroostook County*

Loren F. Carter, M.D.

Waterville

### *Cumberland County*

Roland B. Moore, M.D.

Annandale, Virginia

C. Earle Richardson, M.D.

Brunswick

Ralph E. Williams, M.D.

Freeport

### *Lincoln-Sagadahoc County*

DeForest S. Day, M.D.

Wiscasset

### *Penobscot County*

Henry C. Knowlton, M.D.

Bangor

### *Waldo County*

Allan R. Cunningham, M.D.

Winchester, Massachusetts

DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page 140*

of this vaccine is more difficult in mass campaigns than is the use of the oral vaccine. Furthermore, multiple doses are needed to induce an effective immunity in previously unimmunized persons.

The oral vaccines present a number of advantages. The ease of administration, for example a few drops on a lump of sugar or in a teaspoon of syrup, simplifies mass administration. The recommended dosage schedule is amenable to incorporation into routine pediatric practice. A single dose induces a prompt antibody response to the specific type administered. This vaccine confers a substantial degree of resistance in the alimentary tract to reinfection with wild polioviruses. Thus, herd immunity is a clear benefit of the use of this vaccine. The duration of this herd immunity has not been determined. These properties are, therefore, of special value for organized community-wide immunization programs designed to raise general immunity to the highest possible levels, to reach those segments of the population that have failed to be vaccinated, and to stop epidemics.

Further advantages for epidemic use of the oral vaccine are the promptness of the antibody response to the specific type administered, and the fact that alimentary tract infection with the vaccine strain temporarily interferes with the possible spread of wild polioviruses.

The disadvantages of the oral vaccine include the problem of preservation of the commercial product and the possible seasonal variation in its effectiveness. At the present time the oral vaccines must be stored in the frozen state. After thawing and dilution for use, the presently recommended shelf life is only one week. The disadvantage is slight in community-wide programs, but more significant for general office or clinic practice. Enteric viruses may interfere with the effectiveness of the oral vaccine. Since these are more widely prevalent in the summer months, organized community-wide immunization programs are best undertaken during the late fall, winter and spring months of the year, except in the face of a threatened epidemic.

During the poliomyelitis season of 1962 emphasis must be placed on vaccination of the unimmunized and inadequately protected with one or the other, or

with both of these effective vaccines and also to the initiation of as many well-organized community-wide programs as the supply of vaccines will permit. Individual physicians and health officers will decide which of the two vaccines to use on the basis of their own appraisal of the special factors of their own practice or the circumstances within their own health jurisdictions. Availability of the vaccines may be a determining factor.

D. *Priorities for Use.* During the coming months when supplies of both oral and inactivated vaccines may be limited, the following priorities for use of the poliovaccines are recommended:

1. *Use in Epidemic Control*

2. *Infants.* All communities should organize programs of polio immunization directed to the goal of completing the recommended immunization schedule for all infants by the time they reach their first birthday.

3. *Preschool Children.* Effective programs should be organized to reach all preschool children not yet fully immunized. Completion of the schedule of formaldehyde-inactivated vaccine or initiation of a full course of the oral vaccines may be chosen.

4. *Other Unimmunized Groups.* Older population groups, particularly young adults and parents of small children, should be encouraged to receive immunizations.

E. *Organized Community Use of Oral Vaccine.* Many communities may wish to undertake organized programs to raise the level of poliomyelitis immunity to a maximum with the goal of completely eliminating paralytic poliomyelitis from the community. In such programs all persons in those age groups selected by the community should receive the oral vaccine regardless of past immunization history. The organizers of such programs must be assured that adequate supplies of all three types of vaccine are available before such programs are undertaken.

The immunization schedule sent out to all physicians stresses the continuation of the Salk vaccine in accordance with the recommendations of the American Academy of Pediatrics especially for use for individuals, whereas at the present time, the use of oral vaccine is primarily recommended for community plans in accordance with availability.

# County Society Notes

## ANDROSCOGGIN

March 15, 1962

The Androscoggin County Medical Association met at the Central Maine General Hospital in Lewiston, Maine on March 15, 1962 with 25 members present.

The meeting was called to order by the President, George E. O'Connell, M.D. who introduced James A. MacDougall, M.D., President of the Maine Medical Association.

It was voted to have the Chairman of the Finance Committee look into the possibility of renting a suitable microscope to be loaned to the Poland Community Club for a worthy student.

Mr. Edward J. Carroll of the Internal Revenue Service, guest speaker of the evening, was introduced by Morris Goldman, M.D. A movie "The Inevitable Day" was shown which explained the working of the Revenue Service.

The organization of the Maine District and the functions of its four divisions Administration, Collection, Audit and Intelligence were explained. A lively question and answer period followed.

DONALD L. ANDERSON, M.D.  
*Secretary*

## LINCOLN-SAGADAHOC

May 15, 1962

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at the Ledges Inn in Wiscasset, Maine on May 15, 1962. Sixteen members and guests were present.

The implications of the plan to implement the Kerr-Mills Bill as proposed by Dr. Dean H. Fisher, Commissioner of the State Department of Health and Welfare, were discussed. The society unanimously voted its opposition to this plan as presented.

The secretary was instructed to place advertisements in the local papers informing the public of the television program *Your Doctor Reports*.

GEORGE W. BOSTWICK, M.D.  
*Secretary*

## KENNEBEC

May 17, 1962

The Kennebec County Medical Association held its monthly meeting on May 17, 1962 at the Augusta House in Augusta, Maine. The wives were invited to attend, and after a pleasant social hour and dinner, heard Dr. Gustave H. Todrank, Associate Professor of Philosophy at Colby College, who spoke on "Morals in Medical Practice." Professor Todrank first defined "morals" and then defined "medical practice" to include not only the association between physician and patient but also the care of our society as a whole. He continued his discussion by asking questions which he did not attempt to answer completely but which he offered for consideration by the group.

During the business meeting a resolution was unanimously passed by the Association as follows: That the Kennebec County Medical Association go on record as supporting the

controlled fluoridation of public water supplies as a safe, economical, and effective method of reducing dental caries in children.

EARLE M. DAVIS, M.D.  
*Secretary*

## SOMERSET

May 15, 1962

A meeting of the Somerset County Medical Society was held on May 15, 1962 at the George E. Young Building at the Central Maine Sanatorium in Fairfield, Maine.

A panel discussion on "The Rehabilitation of the Stroke Patient" was presented by The Maine Heart Association. Participants were: Drs. Harold N. Willard of Waterville, John D. Denison of Gardiner and Mr. Leonard Couture, R.P.T.

The Kerr-Mills Bill was discussed.

HARLAND G. TURNER, M.D.  
*Secretary*

## New Members

### CUMBERLAND

Jaime Goldfarb, M.D., Box C, Pownal

### KENNEBEC

Harald J. Schwarz, M.D., Sisters Hospital, Waterville

### SOMERSET

Selic Soroka, M.D., 39 High Street, Skowhegan

## Addition to 1962 Roster

### HANCOCK

J. B. Leith Hartman, M.D., Southwest Harbor

## Deceased

### WASHINGTON

Samuel R. Webber, M.D., Calais, May 30, 1962

## Change of Address

### ANDROSCOGGIN

Cyprien L. Martel, Jr., M.D.

From — 355 Pine Street, Lewiston

To — 91 Bartlett Street, Lewiston

### CUMBERLAND

Robert S. Galen, M.D.

From — 131 State Street, Portland

To — 22 MacMillan Drive, Brunswick

### LINCOLN-SAGADAHOC

Deane L. Hutchins, M.D.

From — 69 Townsend Avenue, Boothbay Harbor

To — Health Dept., Univ. of Maine, Orono



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SEARLE

Research in the Service of Medicine

ACROSS THE DESK — *Continued from Page 139*

per instructor, as compared to 2 per instructor in AMA-Approved Schools. While high school graduation or its equivalent is usually claimed as the entrance requirement, the equivalent can mean, according to the article, "that the student has enough money."

Sixteen of these commercial or private schools are listed in the report, and it is estimated that another twenty-five schools are in operation.

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# The Journal of the Maine Medical Association

Volume Fifty-Three

Brunswick, Maine, July, 1962

No. 7

## Myasthenia Gravis And Thymoma

PETER F. LANSING, M.D.\*

### INTRODUCTION

The recent death at the Togus Veterans Administration Center of a patient with myasthenia gravis and thymoma, having unusual aspects has prompted this review. These features were: (1) a long latent period between the removal of the thymoma and the appearance of symptoms of myasthenia gravis, (2) microscopic cardiac involvement, and (3) unexplained sudden death.

### CASE REPORT

This 46 year old man was admitted to the Togus Veterans Hospital for marked dyspnea on January 2, 1962. In 1955 a chest survey x-ray revealed an abnormality. In October 1956 the patient entered another hospital because of an x-ray which was suspicious of a cyst in the chest. There was a cough productive of white material but no symptoms of myasthenia gravis. A thoracotomy on October 18, 1956 revealed a tumor occupying the anterior mediastinum. This was dissected free by careful piece-by-piece dissection. The tumor was broken into at one point and a small amount was spilled but this was immediately recognized and it was felt that the tumor was adequately removed. It was encapsulated. Grossly, the cut surface revealed irregular, multiple nodules of varying sizes. Microscopically, there was an epithelial type of cell which grew in fairly broad sheets with some tendency occasionally to form whorls, suggestive of epithelial pearls. In other areas the cells were arranged around small cystic spaces and spaces containing small capillaries. Also, collections of lymphocytes were noted in small groups between epithelial cell masses. There were large portions of dense, fibrous tissue, which formed thick bands surrounding and separating larger groups of cells. The impression was thymoma.

Starting approximately November, 1960 there occurred an episode of diplopia, lasting at least four weeks. There was also drooping of the left upper eyelid, weakness of the left

fourth and fifth finger and blurriness of vision when looking towards the left. In January of 1961, a neurological consultant noted ptosis, enophthalmos, inconstant myosis of the left eye, weakness of the interossei of the fourth and fifth fingers, and marked weakness of the abductor digiti quinti. Osteoarthritic changes of the cervical vertebrae were found. A neostigmine bromide test for myasthenia gravis was negative.

The patient developed a viral infection in October and again in November of 1961 associated with vomiting. A chest x-ray in November revealed increased markings at the right base. On December 16, 1961, the patient saw his physician because of marked dyspnea and inability to keep his head upright. At that time the vital capacity was 2 liters.

Other symptoms consisted of distress referred to the lower dorsal spine, radiating upwards to the cervical vertebrae; lameness of the right hand; tendency for the hands to fall asleep; vague, anterior chest distress for several months; and difficulty in getting the hands over the head. There was lessening of activity for three months. The patient experienced no difficulty in swallowing, although there was some vague discomfort in the neck after eating. Myasthenia gravis had not occurred in patient's family.

*Physical Examination* Revealed a moderately dyspneic man. Blood pressure was 122/80. There was a scar over the anterior chest. Chest expansion was  $\frac{1}{2}$ ", lungs were not remarkable on physical examination. Other findings consisted of an unsustained right ankle clonus, difficulty in raising the hands over the head, and keeping the head upright.

The white count was 12,400 with 78% neutrophils, 22% lymphocytes. The sedimentation rate was 33 mm/hr. Urinalysis was normal. Hematocrit was 47%.  $\text{CO}_2$  content was 40 mEq. per liter. Serum sodium, potassium, chlorides, calcium, and phosphorus were normal. Repeat white counts ranged from 9,800 to 13,150 with a shift toward the left. Serial serum  $\text{CO}_2$  content determinations were elevated. The sputa contained *Neisseria* on two occasions. Tests for thyroid functions were inconclusive because of the use of potassium iodide as an expectorant.

The chest x-rays between January 1 and January 17 revealed gradually increasing areas of bronchopneumonia. All areas of bronchopneumonia were improved by January 22nd.

\*From the Medical Service, Veterans Administration Center, Togus, Maine

Electrocardiogram revealed sinus tachycardia.

Pulmonary function studies on January 3 showed a total vital capacity of 2.1 liters, predicted 3.6 liters, 57.2% of predicted. Maximum breathing capacity was 62.5 liters, predicted 106 liters, 59% of predicted. First second vital capacity was 1.4 liters, 66.7% of total vital capacity. There was no dyspnea on climbing one flight of stairs but dyspnea on two flights of stairs. The diaphragms moved equally, but very slowly, over a narrow area of  $1\frac{1}{2}$  interspaces on fluoroscopy.

On January 8th, 1.5 mg. of neostigmine bromide with  $\frac{1}{100}$  grains of atropine was given I.M. Serial vital capacities were measured at 15 minute intervals. The initial baseline value was 1.7 liters, first second vital capacity was 1.2 liters. One-half hour after administration of neostigmine bromide the total vital capacity had increased to 2.7 liters, the first second vital capacity to 2.1 liters. By the following morning the total vital capacity had decreased to 2.1 liters, first second vital capacity to 1.5 liters, and the same afternoon the total vital capacity had further decreased to 1.6 liters, first second vital capacity to 1.4 liters. At that point, a repeat neostigmine bromide test revealed similar improvement. Also, the patient was given a barium swallow before and after neostigmine bromide. There was retention of barium in the valleculae and pyriform sinuses prior to the test and perfectly normal swallowing afterwards.

The bronchopneumonia was initially treated with Chloromycetin,<sup>®</sup> later with penicillin, novobiocin and finally with erythromycin. Except for temperature elevations on the fourth and twenty-third hospital day, the patient was afebrile.

After the diagnosis of myasthenia gravis was made, the patient was placed on neostigmine bromide, 15 mg. at first every four hours and later every three hours. Later pyridostigmine bromide (Mestinon<sup>®</sup> Timespan<sup>®</sup>) 180 mg. four times a day was used. With the development of an upper respiratory infection, and later skin eruption due to novobiocin, neostigmine bromide 15 mg. every three hours was restarted and later changed to pyridostigmine bromide (Mestinon<sup>®</sup>) 60 mg. every three hours.

Five days prior to death, there developed paresis of dorsiflexion of the hands and drooping of the third through the fifth fingers of the right and left hands, and nocturnal dyspnea requiring oxygen. On January 30th the patient became more dyspneic and that evening the pyridostigmine bromide dose was increased to 75 mg. every three hours.

At 6:45 A. M. on January 31st the patient became cyanotic and dyspneic while sitting in a chair. He was given oxygen. Respirations ceased and mouth-to-mouth breathing was attempted. By 7:45 A. M. the patient was placed in a respirator and an intratracheal tube was inserted. External cardiac massage was of no avail. The patient died at 8:45 A. M.

Autopsy performed  $2\frac{3}{4}$  hours after death revealed several small pigmented nodes and two firm, grayish nodules in the fat occupying the anterior mediastinum. Adherent to the hilum of the right lung and the reflection of the pericardium was another irregular nodule measuring 2 cm. in diameter. The right lung weighed 340 grams, and the left lung 240 grams. The lower lobes were finely nodular. There were no areas of consolidation. Mucopurulent secretions were present in the right lower lobe bronchus. The thyroid contained a grayish brown adenoma 1.5 cm. in diameter.

Microscopic examination revealed thymoma in the nodules in the anterior mediastinum consisting of a predominating epithelial-like cell with prominent nucleoli, an oval nucleus, and pale eosinophilic staining cytoplasm. There were intermingled lymphocytes and in some areas lymphocytes surrounded blood vessels. The lungs revealed fibrosis and atelectasis. Within the areas of fibrosis were macrophages containing lipid material. Rare foreign body giant cells and focal collections of lymphocytes were noted. In other areas there occurred thickening of the walls of the bronchioles and a

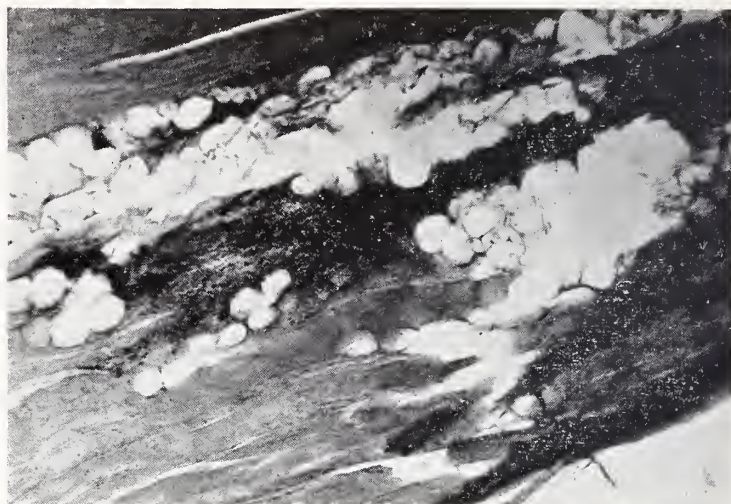


FIG. 1. Skeletal Muscle

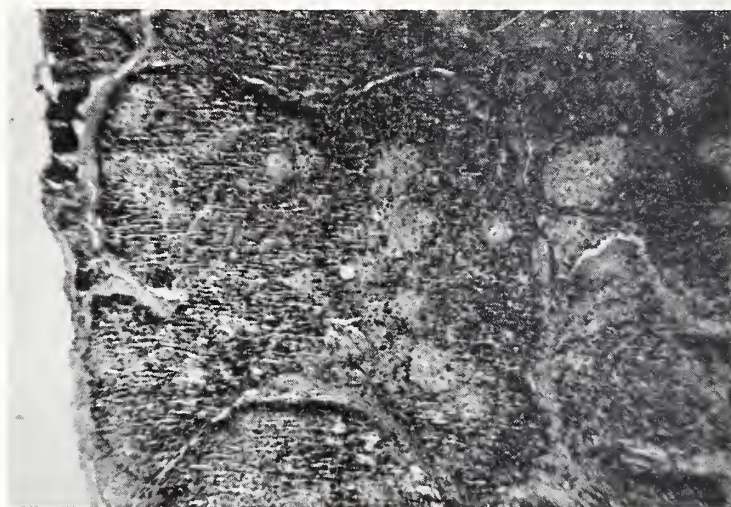


FIG. 2. Thymoma

purulent exudate within the lumen. Minimal, acute bronchopneumonia was present. The myocardium revealed rare, focal areas of lymphocytes with local degeneration of muscle fibers. Sections from pectoral, intercostals, and psoas muscles revealed scattered lymphorrhages. *Escherichia coli* was cultured from secretions of the lower lobe of the right lung. Illustrations of lymphorrhage of skeletal muscle, and a representative section of the thymoma are shown in photomicrographs 1 and 2.

## DISCUSSION

Myasthenia gravis is a chronic disease, characterized by: (A) Variable degree of weakness following the use of various voluntary muscles; (B) A higher incidence in females; (C) A predilection for muscles innervated by cranial nerves, especially extraocular muscles; (D) Increased weakness on voluntary effort, with rapid recovery on rest; (E) Residual weakness, not responsive to rest or to anticholinesterase medication; (F) Dramatic spontaneous remissions; (G) Transient neonatal myasthenia accompanying cases of maternal myasthenia (average duration three weeks after birth, responsive to anticholinesterases); (H) Association with thyroid abnormalities (hypo- and hyperthyroidism).<sup>1,2</sup>

In one-fifth of the patients, the disease remains limited to the extraocular muscles. In others, there is steady progression over periods of weeks, months, or

years. Most patients fall into an intermediate group, whose course is initially one of progressive involvement, reaching a plateau within the first year, after which there occur periodic exacerbations and remissions. Occasionally, remissions are complete and prolonged; usually they are temporary and partial. Extraocular muscle weakness usually is the first sign of exacerbation, and the last sign of remission.

Exacerbations occur with upper respiratory, and other infections, emotional factors, hypo- and hyperthyroidism.<sup>2,3</sup>

Most women feel weakest for several days to two weeks prior to the menstrual period.

Remissions are associated with pregnancy in a variable manner. In most instances, there are moderate relapses during the first trimester, followed by a partial or complete remission. The course in the post-partum period is variable.<sup>4,5</sup>

Remissions are also noted during the induced euthyroid state.<sup>2</sup>

Death occurs in 85% within six years of onset.<sup>6</sup> Overall, the menstruating female lives longer, while both sexes after the age of 50 have a much worse prognosis. Over the age of 50 the predominance in females disappears and males are seen more frequently.<sup>1,7</sup>

The presence of thymoma has usually an adverse effect on both life expectancy and the severity of the myasthenia gravis.<sup>1,7,8</sup>

Thymoma is associated with a male preponderance and occurs later in life.<sup>1</sup> In some instances, the detection of a thymoma precedes the onset of myasthenia gravis by many years. In at least three instances myasthenia gravis has appeared several years following apparent total thymectomy.<sup>3,9,10</sup>

In our patient no symptoms of myasthenia gravis were present at the time of surgery in 1956. The operative notes stated that the tumor was broken into at one point and a small amount of tumor was spilled but this was immediately recognized and it was felt that the tumor was adequately removed. The symptoms of myasthenia gravis did not appear until 1960.

Obviously the facts in this case do not permit a definite conclusion as to whether the thymoma found at autopsy represented growth of tumor left behind at the original operation, or was the development of an entirely new lesion. In either event, the occurrence of myasthenia gravis four years after the removal of the thymoma is most unusual.

Histologically, thymomas fall into four groups: (A) Tumors composed of thymic lymphoid elements; (B) Tumors of epithelial elements comprising Hassall's corpuscles which grow as essentially epithelial structures, resembling squamous cell carcinoma; (C) Tumors composed of spindle cells resembling fibromas or fibrosarcomas; and (D) Tumors of primitive reticular epithelium growing as fibromyxomatous masses.<sup>12</sup> Mixtures of the above elements take place. Sometimes there are perivascular cuffs of lymphocytes reminiscent of the

perivascular lymphocytic accumulation in experimental autoimmune disease.<sup>1</sup> The correlation between histological and clinical malignancy is poor.<sup>11</sup> Thymomas associated with refractory anemias due to erythroid hypoplasia, granulocytopenia, thrombocytopenia and acquired agammaglobulinemia have been reported. Some of these patients with cytopenias also had myasthenia gravis.

There is much controversy over the value of thymectomy in patients who have myasthenia gravis without thymomas. Most observers agree that surgery may be of benefit in the female;<sup>1,11</sup> however, it is most difficult to come to any valid conclusion when it is well known that the disease is most benign in the female who is of child-bearing age.

In recent years, experiments have suggested that the thymus is an immunological rather than an endocrine organ. In the immature animal the thymus is necessary for the production of circulating antibodies to foreign proteins. The adult thymus has not been demonstrated to produce antibodies in response to remote injections of antigen, but is immunologically reactive to the direct injection of antigen, producing intense germinal center formation similar to that seen in the myasthenic thymus. The thymus is also a source of circulating lymphocytes and lymphocyte stimulating factor.<sup>1</sup>

Myasthenia gravis is associated with electrophysiological abnormalities consisting of a decreased acetylcholine effect on the motor end plate. There is disagreement as to its cause. The two major possibilities are (a) diminished synthesis or release of acetylcholine by the nerve endings, and (b) decreased sensitivity of motor end plates to acetylcholine liberated in normal amounts.

Increased destruction of acetylcholine has been reasonably well excluded.

Recently, Dahlback et al<sup>13</sup> have obtained recordings from myasthenic motor end plates in vitro. Miniature motor end plate potentials (which result from the spontaneous release of packets of acetylcholine from nerve endings) were found to be of normal amplitude indicating no decrease in motor end plate responsiveness. The frequency of release of the packets of acetylcholine was less than normal. This evidence weighs heavily in favor of a defect in nerve endings. However, as these muscles were not clinically involved, one still could not rule out insensitivity of the motor end plates of the clinically involved muscles.

Anatomical changes in the muscle fibers involved by myasthenia gravis consist of (a) acute coagulative necrosis, and (b) lymphorrhage. At least one author considers the latter to be secondary to necrosis of muscle fiber. However, this has also been seen in pericapillary and perivenular locations, which is characteristic of experimental auto-immune disease.<sup>1</sup> In the myocardium, isolated lymphorrhages have been observed several times with or without slight alteration of muscle fibers.<sup>5</sup> Such changes occurred in our patient.

Electron microscopic studies have revealed abnormalities in the sarcolemma, synaptic clefts and axoplasm of the nerve terminals.<sup>1</sup> Immunological abnormalities have been described. Fluoresceinlabeled serum globulin was used. The serum globulin in some patients was bound to skeletal muscle; the binding was associated with fixation of complement. The bound serum globulin was a 7S gamma globulin, which was found in certain regions of the muscle fiber. It was capable of binding to normal skeletal muscle, but not to human cardiac muscle.<sup>1</sup>

Sudden death may occur in myasthenia gravis. Patients who do not appear to be in danger at one moment may become cyanotic and mentally obtunded the next. Neostigmine bromide, the use of a respirator, and aspiration of pulmonary secretions may at times be of value, but at other times, death occurs in an inexplicable manner.<sup>5</sup> The sudden collapse of our patient would fall in this category. It has been suggested that exertion may cause sudden respiratory weakness in an obscure manner, possibly through release of a curare-like factor.<sup>5</sup>

The association of myasthenia gravis with hypo- and hyperthyroidism has been mentioned.<sup>2</sup>

Drugs used in therapy are outlined in Table 1.<sup>14</sup>

<i>Drug</i>	<i>Preparation</i>	<i>Equiv. Dose (in Mg.)</i>	<i>Dose Interval</i>
Neostigmine bromide (Prostigmine)	15 mg. tablets	15 mg.	2-4 hours
Pyridostigmine bromide (Mestinon)	60 mg. tablets	60 mg.	3-6 hours
Timespan Mestinon	180 mg. tablets	60 mg.	8-12 hours
Ambenonium (Mytelase)	10 and 25 mg. tablets	5 mg.	6-8 hours

The patient should start at a dose level adjudged to be inadequate. At intervals the dose is increased and strength measurements are made, preferably of those muscles that are the most severely affected. In our patient, respiratory muscles were most severely affected and doing serial vital capacities was of great value in judging effectiveness of medication. When a given dose produces no further increase or even decrease in strength, the dosage should be reduced. It is preferable to use neostigmine bromide or pyridostigmine bromide during the day and the prolonged acting form of pyridostigmine bromide, (Timespan Mestinon) at night time. Generally, neostigmine bromide 15 mg. is given at three hours intervals and pyridostigmine bromide (Mestinon) at four hour intervals. To counteract the cholinergic effect of the drugs, atropine grains 1/200 to 1/100 can be given 20 minutes before each troublesome dose (usually this is the morning dose).<sup>14</sup>

The patient with impending respiratory failure should be placed in a respirator. As an indication of the need for a respirator, an absolute value of vital capacity is not especially useful. Of much greater value are inability to clear secretions and inadequate respiratory excursions. Tracheotomy is strongly advisable, once

respirator therapy is necessary, in order to remove secretions adequately.<sup>11</sup>

Crises in myasthenia gravis fall into two chief groups: (A) Myasthenic, when due to relapse of myasthenia. These crises are characterized by profound weakness, abnormal fatiguability of muscles of respiration, as well as those of cranial nerves. (B) Crises due to overdosage with cholinergic drugs, (neostigmine bromide, pyridostigmine bromide, etc.). These crises have all the symptoms of the myasthenic crisis, but in addition have (a) muscarinic manifestations with excess salivary, sudorific, lacrimal and bronchial secretions, miosis, anorexia, nausea, abdominal cramps, diarrhea, incontinence of bowel and bladder, dyspnea, substernal pressure and pulmonary edema; (b) nicotinic symptoms with fasciculations, muscle spasm, and weakness; (c) central nervous system symptoms with anxiety, restlessness, vertigo, headache, confusion, coma, and convulsions.<sup>11</sup>

Horenstein has emphasized the presence of fasciculations of the small muscles of the face and hands as useful criteria for overdosage.<sup>7</sup>

The Edrophonium (Tensilon®) test may be of value. One cc. (10 mg.) of Tensilon is placed in a tuberculin

syringe; 0.2 cc. of this is injected intravenously during a period of 15 seconds and the needle is left in place. If no reaction occurs after 30 seconds the remaining 0.8 cc. are injected. If improvement occurs, the crisis is probably myasthenic, and cholinergic therapy is indicated. If fasciculations and/or no improvement follows the injection of Tensilon, the crisis is probably of cholinergic type.<sup>11</sup> Some consider this test too hazardous because of the occasional occurrence of cardiac arrest.<sup>7</sup>

Much needs to be learned about myasthenia gravis and the thymus. Myasthenia gravis may be a unique type of response to some, as yet unknown, immunological abnormality. The well-documented thymic abnormalities, the few cases in which myasthenia gravis has developed following apparent total thymectomy, all indicate our great ignorance and challenge to future investigators.

SUMMARY

A patient with myasthenia gravis is described who had a thymoma resected in 1956 and who developed symptoms of myasthenia gravis in 1960. At the Togus Veterans Hospital the patient presented himself chiefly with symptoms of marked respiratory failure and weak-

ness of neck muscles. Death occurred suddenly in an unexplained manner.

The literature is reviewed with reference to myasthenia gravis and thymoma.

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## Volvulus Of The Small Bowel - With Case Report

G. G. BROAD, M.D.\*

Volvulus of the small or large intestine is one of the causes of intestinal obstruction. Volvulus of the small bowel occurs in the United States in 2-6% of all cases of intestinal obstruction, according to several reported statistics.<sup>4,5,11</sup> In the large bowel the sigmoid colon is one of the areas relatively prone to volvulus. Combinations of large and small intestine volvulus (that is, the right colon and adjacent small bowel) are not infrequently found in infants and children associated with congenital anomalies of rotation of the mid-gut. In India, Africa, Serbia, Russia, Sweden and Finland, it is reported that volvulus in general occurs more frequently than in other regions, probably due to a bulky farinaceous diet.<sup>16,17</sup>

#### ETIOLOGY

Volvulus is a twist or rotation of a section of intestine. Cohn and Blaisdell<sup>1</sup> state that two conditions are necessary for a volvulus to occur, namely a fixed point about which the bowel may rotate, and a sufficiently long mesentery to permit the twisting motion to develop. In infants and children incomplete rotation of the mid-gut (end of duodenum to mid-transverse colon), which is an embryologic defect, may develop about the superior mesenteric artery as the fixed point. The unattached or incompletely attached mesentery of the mid-gut permits undesired mobility of the same section of intestine. Volvulus may readily develop under these conditions. A very interesting review of various anomalies of failure of rotation of the mid-gut is given by Bennett<sup>2</sup> in the Medical Journal of Australia. Gross,<sup>6</sup>

of our country, describes the surgical treatment. Williams et al<sup>19</sup> report that failure of normal mid-gut rotation is found in adults by X-ray studies only once in 20,000 cases.

In addition to the faulty bowel development, noted especially in infants and children, the volvulus in adults is found in sigmoid colons which have a long loop, cecums unattached to the abdominal wall, transverse colon, small bowel and stomach, in that order of frequency, according to Cohn and Blaisdell.<sup>4</sup> It seems reasonable that a long mesentery is a pre-requisite for volvulus; however, Rose<sup>11</sup> in 1956 reported a series of 39 cases of small bowel volvulus in all of which the mesentery was normal. In this series there were 16 cases of unknown cause for volvulus, 8 cases of volvulus due to congenital bands and 15 cases of volvulus due to acquired adhesions. A number of authors reporting cases of volvulus indicate that a post-operative status following various kinds of surgery precedes the onset of volvulus in a significant percentage of cases.<sup>3,5,11,13</sup> Adhesions, post-operative, seem to be associated with volvulus as the most frequent post-operative causative factor. Turner and Sloan<sup>14</sup> report volvulus developing in a patient after gynecological surgery. During the operation, the small bowel was several times packed off into the upper abdomen. The patient's symptoms of volvulus began on the second post-operative day. She required surgery for obstruction (volvulus) during which procedure the twisted small bowel was found to be the cause of the obstruction. It was noted that at her first operation the small bowel had not been removed from its "packed off" and perhaps twisted

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position at the time of closure of the abdomen. Other authors<sup>9</sup> suggest that the small bowel may be twisted on itself inadvertently during manual explorations through a small abdominal incision. Gannon and Harrington<sup>5</sup> reported some cases without any evidence of etiological factors being present; see Rose<sup>11</sup> also.

#### SYMPTOMATOLOGY

Volvulus is one type of intestinal obstruction. The symptoms are therefore those of obstruction, pain, nausea, vomiting, distention. Peristalsis may be diminished to absent. There was abdominal tenderness, usually with spasm or rigidity of the abdominal wall, in 43% of one group of 31 cases,<sup>13</sup> rebound tenderness in 26% and a palpable mass in 13% of the same 31 cases.

Volvulus may be represented by a recurring series of mild symptoms which Smith and Perry<sup>13</sup> found in 60% of their cases. Blood studies are not characteristic. The same physicians reported an average white count of 11,300 in 39 cases of volvulus.

#### DIAGNOSIS

The diagnosis of volvulus is difficult. Correct pre-operative diagnosis was made in 9 of 31 cases reported by Smith & Perry,<sup>13</sup> in 4 of 23 cases reported by Gannon and Harrington<sup>5</sup> (discussion) and in 7 of 36 cases by Moretz and Morton.<sup>9</sup> The difficulty in some instances is to distinguish volvulus from other types of intestinal obstruction. At other times the non-specific character of the symptoms and findings does not bring the disease entity to mind. The presence of shock has been reported as a feature which is associated with gangrene of the twisted bowel.<sup>12,18</sup> Rose<sup>11</sup> too, reported shock present at some time in 8 cases, of which 3 died of shock, primarily.

X-ray studies (abdominal flat plate) have been occasionally reported in volvulus cases to show no abnormality of the intestines, but more commonly there are distended loops of the small intestine (or of large bowel in colon cases). An enormously distended loop suggests volvulus. Another suggestive X-ray feature is a loop filled with gas and fluid which is longer than that usually found in intestinal obstruction. Ryan,<sup>12</sup> a radiologist, also mentions that the curved lines of the walls of the affected bowel converge towards the site of the volvulus. In addition he feels that a barium enema may be necessary, and if this is so, there is little risk in this procedure. A different kind of X-ray study is called pneumoperitoneography. Perry, VonDrashek, and Wangenstein<sup>10</sup> have reported studies made by injecting 500-1,000 cc's of air into the peritoneal cavity when a strangulated loop of bowel is suspected. These writers have found that the strangulated bowel has an increased amount of blood in the lumen and in the wall of the bowel, which increased density allows the bowel loop to stand out clearly against the background of air in the rest of the abdominal cavity. In their studies, infarction due to arterial or venous block alone

did not give this clear picture of the strangulated bowel. These authors reported 3 case histories of patients with strangulation of the bowel diagnosed by this X-ray technique.

The diagnosis of volvulus is suggested, therefore, if intestinal obstruction is indicated by the case features, with either unusually large diameter to the distended bowel as seen by X-ray, or an unusually long loop of distended small bowel with fluid level therein. Recurring episodes of abdominal pain with vomiting may suggest volvulus. The additional features of shock which may be associated with gangrene or perforation of the bowel, may be present, in which case abdominal rigidity is very likely to be noted also.

#### TREATMENT

Volvulus usually requires surgical correction. Recurring episodes of mild degrees of volvulus will probably come to surgery. The more severe the degree of torsion of the bowel, the greater the risk of strangulation of the blood supply. Gangrene of the twisted loop of bowel occurred in 8 of 47 cases reported by Christensen.<sup>3</sup> A number of other isolated case reports mentioned gangrene of the affected loop.<sup>5,15</sup> It is the progressive interference with the blood supply which increases the hazard of volvulus in addition to the element of intestinal obstruction.

Abdominal exploration will reveal the distended loops of bowel. Constricting or obstructing bands must be severed. The torsion of the bowel and mesentery must be untwisted. Several writers on this subject indicate that for the twisted bowel, with bowel viability still present, simple untwisting of the volvulus is all that is needed.<sup>8,11</sup> Wangenstein<sup>17</sup> agrees and also has a suggestion, namely, fastening the mesentery of segments of the small bowel to the posterior abdominal wall, in a fanwise manner, to reduce the chances of recurrence. Rose<sup>11</sup> has reported a series of 39 cases of volvulus of the small bowel treated only by untwisting the mesentery with no recurrences.

#### MORTALITY

Relatively high mortality rates are reported, 13.8%-36%.<sup>11,12,13,17</sup> Most fatal cases mentioned by these authors were those in which gangrene, with or without perforation, had developed. These facts indicate the need for increasing accuracy of diagnosis, and earlier surgery.

#### CASE HISTORY

A forty-year-old male was admitted to the V.A. Hospital, Togus, Maine, 10/13/61. During the past 2-3 months he had had repeated episodes of vomiting, which recurred so frequently that most of his food had been regurgitated. One week prior to admission he began to have epigastric pain and a burning sensation, referred to the left chest and shoulder, (he has had several admissions to this hospital for a duodenal ulcer since 1953). Twenty hours before admission to the hospital, on this occasion, lower abdominal pain suddenly appeared, which developed with increasing severity. He had four loose, small bowel movements at the onset of the

pain and vomited several times since then. He had not had hemoptysis or blood in the stool.

**PHYSICAL EXAMINATION:** Patient was a well-developed and nourished adult male in acute distress, with nausea, retching, and abdominal pain. He was unable to lie quietly in bed. His heart and lungs were normal. The abdomen was slightly distended. The rectus abdominus muscles were moderately rigid but relaxed in some degree on deep breathing. There was generalized abdominal tenderness with a moderate degree of rebound tenderness in both lower quadrants. Dullness was present in the left lower quadrant. Peristaltic sounds were absent. Rectal examination revealed a small amount of semi-solid stool without blood. There was no rectal tenderness.

**LABORATORY:** WBC 14,700, polys, 87%, Serum amylase, 80 units, Serum Bilirubin 0.55 mg%. Urine showed 2 plus albumin and many WBCs. X-ray (abdominal flat plate): On admission there was no evidence of free air within the peritoneal cavity. There was a ground-glass appearance to the abdomen, indicating either intra-peritoneal or intra-intestinal fluid. There were multiple moderately distended loops in the small bowel with air-fluid level seen in the decubitus film. There was a single markedly dilated loop of small bowel in mid-abdomen. No large bowel gas was seen except a small amount in the cecum.

**X-RAY DIAGNOSIS:** Small bowel obstruction probably at mid-ileum.

Because of the history of peptic ulcer, a tentative diagnosis of perforated ulcer was made. Operation was undertaken within a few hours after the patient's admission to the hospital.

**OPERATION:** A right mid-abdominal para-median incision was made. Distended loops of small bowel were immediately noted. They were blue in color, the color of blue sky. They were filled with air and fluid. There was a moderate amount of free fluid in the peritoneal cavity. The bowel surfaces were slippery and the free fluid was slightly soapy in appearance. The distended loops of the small bowel were brought out beyond the abdominal wall. It was seen that there was a twist in the mesentery in a clockwise manner with both distended and collapsed loops of small bowel intertwined in a confusing mass. So confusing was this that it took several efforts to disentangle the loops even after the initial detorsion of the mesentery had been accomplished. By following the small bowel from the origin of the jejunum at the ligament of Treitz all the way down to the ileo-cecal junction, the twist in the mesentery was straightened out without difficulty. Immediately the incomplete blood flow was relieved and the color of the bowel improved to normal. Considerable amounts of gas and fluid were expelled up the Levin tube in the stomach. An estimated 6-10 feet of the midsection of the small bowel were involved in the twisting process. The mesentery of the small bowel seemed long. Approximately in the middle of the involved bowel was seen a large Meckel's diverticulum with a wide patent lumen into the bowel, which was distended along with the rest of the small bowel. Because of acuteness of the volvulus, no surgery for the Meckel's diverticulum was undertaken. It was then possible to inspect the stomach. A scarred area of the duodenal region was present 1-2 cm. in diameter. There was no perforation or inflammation of this scarred area. After the loops were straightened out, the abdomen was closed with interrupted sutures. The post-operative course was essentially uneventful. Paralytic ileus was present for several days. A few bowel sounds were present on the third post-operative day. Recovery was satisfactory from there on. The patient was discharged on the 13th post-operative day, with the wound healed, bowels functioning and the patient feeling reasonably well. The date of discharge from the hospital was 10/26/61. He was re-admitted 11/20/61 for diarrhea, vomiting and abdominal pain of 17 days' duration. At

this time, the abdomen was generally tender. Bowel sounds were normal. The day after this admission the abdomen was flat and soft without masses or tenderness. Hyperperistalsis was present. Abdominal flat X-ray plate showed normal gas in the large bowel and no abnormal small bowel gas. Diarrhea and vomiting ceased after two days in the hospital. Bowel function returned to normal and the patient felt improved. He was in the hospital for nine days. He was discharged 11/29/61 with a final diagnosis of Enteritis, acute.

It is possible that the patient had a mild recurrence of his volvulus. If so, we shall probably see him again.

#### SUMMARY

Volvulus of the small bowel is discussed. The difficulty in diagnosis is apparent in a case report. The high mortality rate reported in several series of cases in the past few years warrants an increased awareness of this possible diagnosis and earlier surgical intervention.

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# Simple EKG Ruler

PETER F. LANSING, M.D.\*

It has been found by this writer that the rapid reading of electrocardiograms is materially aided by the use of a simple homemade ruler which is described in the following note.

It is with some trepidation that this simple device is introduced in this computer oriented age. Presbyopia, myopia, and advancing age were the godparents.

The device gives the following information:

- 1. The PR interval is less or greater than 0.20 seconds.
- 2. The QRS interval is less or greater than .08 seconds.

- 3. The QRS interval is less or greater than .12 seconds.
- 4. The QT interval is less or greater than .36 seconds.
- 5. The ST segment is elevated or depressed.
- 6. The heart rate can be calculated.

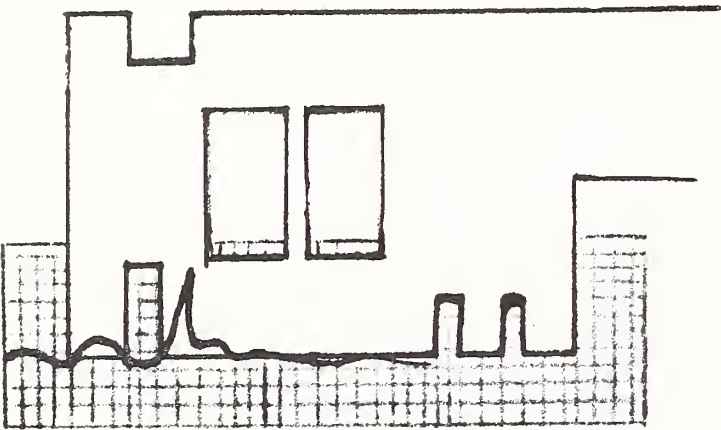


FIG. 1

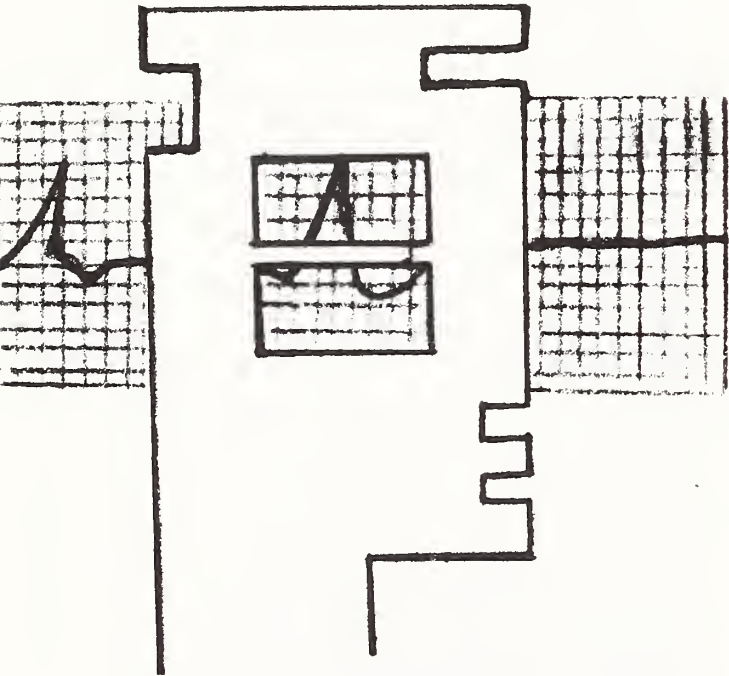


FIG. 2

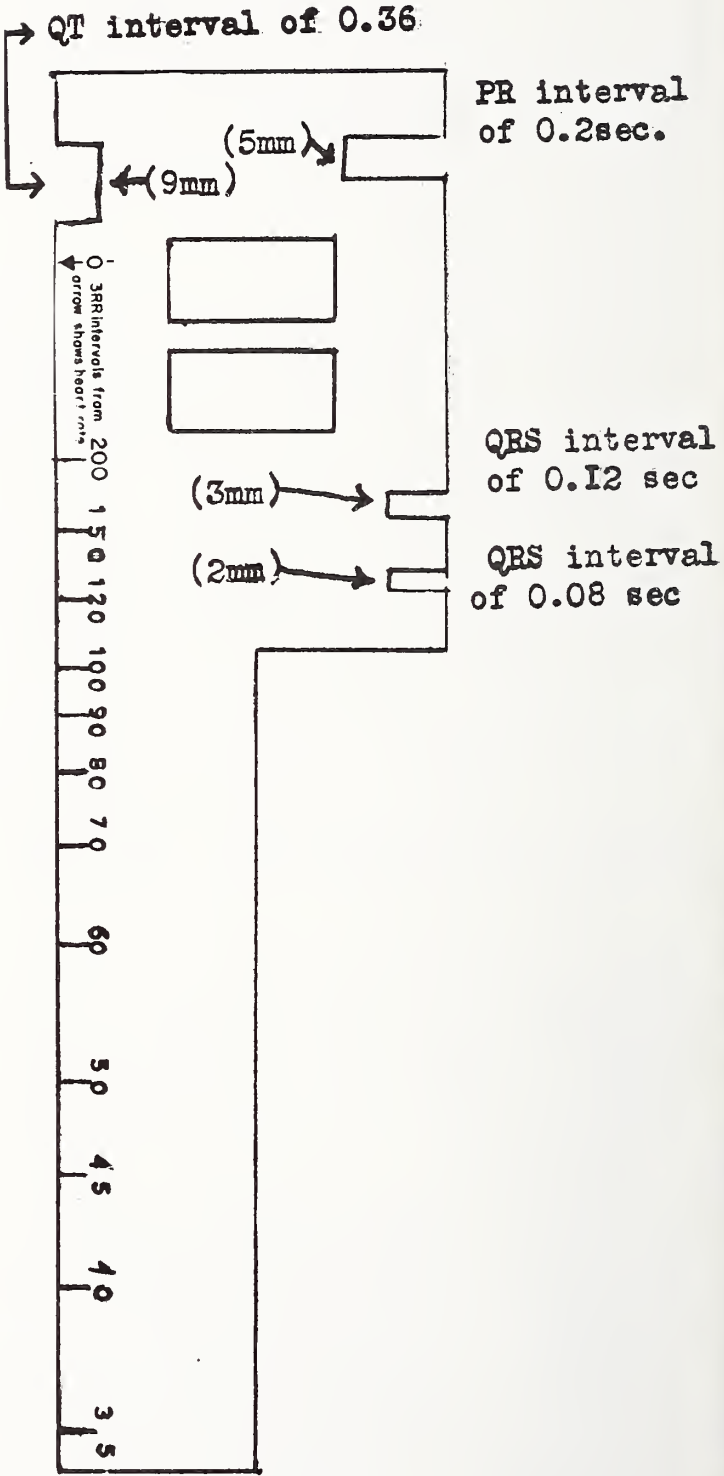


FIG. 3

\*From the Medical Service, Veterans Administration Center, Togus, Maine

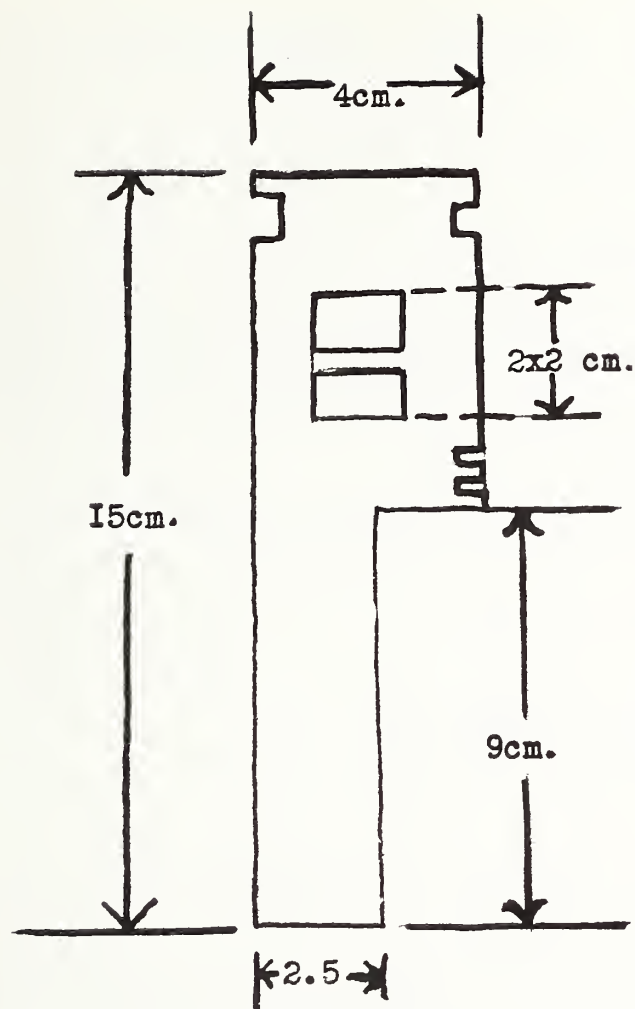


FIG. 4

The notches of the ruler are used to measure the PR, QRS, and QT intervals. An illustration of measuring the PR interval is shown in figure 1. The QRS intervals and QT intervals are measured in similar fashion. Obviously, the PR and QT intervals are rate dependent and for these the device is used for screening purposes.

The window in the center is used for determining elevation or depression of the ST segment, as shown in figure 2. The left side of the bar across the center window is placed just above the PR interval, leaving the right side of the bar to determine if the ST segment is elevated or depressed. The heart rate is measured by means of the divisions at the side of the handle, as seen in figure 3.

The ruler is made of sheet aluminum (which has held up well since September of 1961). The specifications are given in figures 3 and 4. Figure 3 shows the device in actual size. It can be photographed and very easily reproduced by any machine shop.

I am greatly indebted to Mr. Manley F. Cates for his suggestions and drawings and to Mr. Nelson Huntsman of the Occupational Therapy Division, for giving substance to this idea.

SUMMARY

A new simple ruler is described which measures pre-determined, significant, PR, QRS, and QT intervals, determines elevations and depressions of ST segments, and measures heart rate.

THE UNEXPECTED IN NEW DRUG RESEARCH

The most challenging problems in the field of drugs center mainly on diseases which are not yet clearly understood. The research efforts required for their resolution will entail basic understanding and a close integration of many disciplines. The problems still with us are difficult and complex. No one can state with certainty what the future holds. Indeed, in the field of research and new products we are often puzzled — or even astonished — at the least expected point. — Dr. Ernest H. Volwiler, former president, Abbott Laboratories, to Senate Subcommittee on Antitrust and Monopoly, Dec. 9, 1961.

# The Journal of the Maine Medical Association

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## Across The Desk

### **Prospective Control Of Hospitals In Quebec (Too Close For Comfort)**

Editors Note: At times we are inclined to think that tomorrow is "soon enough," and that the medical problems of other countries are not our problems. The following letter should make us realize that every day we delay taking action against those aspects of "Big Government" to which we sincerely object, is in reality, aiding the cause of "Big Government" and hastening the day when we will all live under its rigid control.

Any bill that is drafted leaves the control and regulation of its operation in the hands of a Secretary or his designates. It is the regulation as interpreted by someone quite remote, that will determine how, where and when we treat our patients.

To: Editor, Journal of the Maine Medical Association

From: Paul A. Webb, Executive Director

Associated Hospital Service of Maine

Date: June 18, 1962

The Quebec government November 11, 1960 proposed in the legislative assembly a hospital insurance plan for all residents of the province, starting January 1, 1961. The day following the introduction of this legislation the VANCOUVER SUN said:

"Quebec's hospitals, 85% of them administered by Roman Catholic religious orders, would retain full autonomy in the hiring of nursing and medical staffs and in the formation of medical bureaus."

The legislation was passed and subsequently implemented.

Approximately 18 months later the Quebec Legislative Assembly was considering "Bill 44" concerning

which THE GAZETTE (Montreal) of May 19, 1962, reported as follows:

### **ABSOLUTE CONTROLS SET FOR HOSPITALS BY BILL PERMIT NEEDED BY ALL PUBLIC AND PRIVATE INSTITUTIONS**

BY HERBERT LAMPERT

Quebec — A Hospitals Bill establishing absolute Government control over medical and financial administration of public and private hospitals in Quebec was given first reading in the Legislative Assembly yesterday.

Excluded are all mental institutions now governed by the Mental Patients Institutions Act — with one exception.

Montreal's L'Institut Albert Prevost will be recognized by the Government as a public hospital covered by the new law, Health Minister Alphonse Couturier told the Assembly in his explanatory remarks.

Under the terms of Bill 44 all public and private hospitals require a permit from the health minister to be issued or renewed annually. It may be revoked by the minister for any reason he deems valid.

Section 22 of the bill gives the cabinet the right to expropriate any hospital whose permit is cancelled, or any hospital which fails to comply with Section 5 within six months after the act is enforced.

Section 5 reads: "A public hospital that is not the property of Her Majesty must be owned by a corporation having no other object than that of maintaining such a hospital.

Only such a corporation may be the lessee, conces-

sionnaire or administrator of a hospital belonging to Her Majesty."

#### CABINET TO MAKE REGULATIONS

The cabinet would have the power to make regulations respecting:

- A) Classification of hospitals for carrying out this act and the definition of a physician's or dentist's consulting room.
- B) The related or ancillary powers that may be exercised by a nonprofit corporation the object of which is to maintain a hospital.
- C) The By-Laws that such a corporation may or must make;
- D) The construction of hospitals, their equipment and staff;
- E) The contents, preservation and examination of medical records and the photographic reproduction thereof;
- F) The conditions on which physicians and dental surgeons are admitted to hospitals there to practice their profession;
- G) The conditions on which students of medicine and of dental surgery are admitted to hospitals to study their profession;
- H) The admission and discharge of patients;
  - I) The care and treatment to be provided to patients;
  - J) The regulations for the medical staff, the medical board and all medical committees;
- K) The disciplinary regulations applicable to patients or visitors;
- L) The accounting and auditing and the reports and statistics to be furnished to the minister and the registers to be kept;

#### FEES TO BE CHARGED COVERED

- M) The form of permits and of applications for permits;
- N) The fees payable by private hospitals;
- O) The provisions the infringement of which constitutes an offense.

The bill provides for the institution by the cabinet of a conciliation committee to settle any irreconcilable dispute between the board of management and medical board of a public hospital. The committee's decision will be final. The bill also establishes that its regulations will prevail over any inconsistent provision of a hospital's charter.

Recommendations of the Chaout and Tellier Commissions following investigation into Jean Talon and Fleury Hospitals respectively are strongly reflected in the bill.

Every public hospital must have a board of management which would include at least one Physician designated by the medical board of the hospital. Representation by the medical or dental profession on the board is limited to one-third of the members and not more than

another third from among others employed at the institution. The chairman cannot be chosen from among the professional group. No member of the board may be chosen from among relations, or associates, to the degree of first cousin.

Each hospital must have a medical board elected by the active medical staff and responsible for medical care and scientific organization of the hospital to the board of management, or in private hospitals to the proprietor.

Finally no one can establish, convert or enlarge a hospital without authorization of the cabinet.

The bill is expected to come up for second reading next week.

#### Inspection Of Physicians' Records By Insurance Agents

The agent of an insurance company appeared in a physician's office with the signed consent of a patient and demanded to photostat pertinent records on a portable machine which he carried with him. Is the physician obligated to turn the records over to the agent on the spot? In the absence of a court decision in the particular state, the following general principles will be of assistance in answering the question:

1. Medical records belong to the physician. On this, the law is pretty clear. They are made by him for his own use and he owns them.
2. The patient, however, has a right to know the medical data relating to his own condition. That is, he is entitled to the information contained in the records but not to the records themselves.
3. The patient can give to the insurance company no more right or interest in the records than he himself has.
4. No person has a right to inspect the actual records at all without the consent of the physician who owns them, unless, of course, a subpoena for them has been properly issued.
5. The physician has a right to insist that he himself make the copies or abstracts of his records and that he be compensated for his time in making them.
6. The physician also has a right to insist that the medical data be furnished at his convenience. That is, he is under no obligation to cancel his appointments and devote all his time, at once, to preparing the requested information.

Summarizing the problem of furnishing copies of records to representatives of insurance companies, Attorney Eugene R. Warren says, "In furnishing medical records and preparing medical reports on behalf of the patient, the physician many times expends considerably more time than the fee which he feels he can appropriately charge therefor justifies. Many physicians consider this work a necessary nuisance solely for the convenience of the patient and as a professional courtesy to the

patient. The physician's place in the scheme of our society is to treat the sick and not to prepare historical reports or explanatory documents. It would thus seem that every effort should be made to minimize the paper work demanded of the physician by the patient which does not directly bear upon diagnosis and treatment of the illness from which the patient is suffering." (Legal Problems Involving Medical Records, Eugene R. Warren, Esq., Legal Counsel, Arkansas Medical Society, *Journal of the Arkansas Medical Society*, November, 1961.) (The Doctor & The Law, Volume V, No. 4)

### 1961 Strides In Medicine Highlighted

An American Medical Association poll of deans of medical schools highlighted advances in medical science during 1961 on most of the major disease fronts. Major strides were recognized in: 1) Knowledge of human genetics — expanding so rapidly that elimination of many serious human diseases seems possible. 2) The search for drugs that will cure or prevent cancer — with significant discoveries. 3) Development and discovery of new pharmaceuticals, particularly the new oral polio vaccine, the measles vaccine, and synthetic penicillin. 4) New knowledge about hepatitis which may eventually lead to a vaccine against the only remaining epidemic disease which is still increasing in the United States. (American Medical Association, 535 N. Dearborn St., Chicago 10, Dec. 31, 1961)

### Labor Lobby Tops Million

The next time someone criticizes the AMA as the "biggest lobby in Washington," present these facts which

have just been uncovered by AMA's Legislative Department . . . During 1961, labor unions spent \$1,024,049.38 for lobby expenses while the AMA's lobbying expense was \$163,404.61 . . . The total for all AFL-CIO unions alone came to \$706,961.79 . . . Of this amount, the AFL-CIO parent group spent \$133,919.01 . . . AFL-CIO affiliates spending large sums were: Farmers' Educational and Co-operative Union of America, \$88,272.56; United Federation of Postal Clerks, \$73,867.83; and the International Association of Machinists, District Lodge No. 44, \$71,736.62. (Legislative Roundup, March, 1962)

### Advance In Fight Against Pneumonia

The "Eaton agent," known to scientists since 1944 and recently shown to be an important cause of pneumonia, has been grown for the first time in a cell-free medium and revealed as a member of an obscure group of microbes called "PPLO" (Pleuropneumonia-like-organisms). This is the first PPLO linked with any human disease. The findings represent not only another step in advancing diagnosis and prevention of a prevalent form of pneumonia but also the "capture" of an agent that may give information about a whole group of organisms. (National Institutes of Health, Bethesda 14, Md., Jan. 23, 1962)

### In Fewer Words

April's recipients of social security checks passed 17 million mark, and nearly 12 million of them were in 65-and-over brackets . . . (Washington Report on the Medical Sciences, No. 776)

## FROM TEST TUBE TO BEDSIDE

Each year the pharmaceutical industry in this country makes, discovers or synthesizes more than 100,000 chemical compounds or substances. Of these perhaps about 2500 to 2800 may reach the stage of investigation to determine their usefulness in humans. And of this latter number perhaps 30 to 40 may eventually appear on the market. Incidentally, to undertake this huge job of searching and testing there are more than 16,000 scientific personnel employed by the pharmaceutical industry. And about 5 to 6 years, as an average, intervene from the time an idea is discovered in a test-tube and when it finally emerges for general use in sick people. — Austin Smith, M.D., in *Military Medicine*, March 1962.



RALPH C. STUART, M.D.

*President, Maine Medical Association*

1962-1963

## Ralph C. Stuart, M.D.

*President, Maine Medical Association*

1962-1963

Ralph C. Stuart, M.D. of Guilford, Maine assumed his duties as President of the Maine Medical Association on June 18, 1962 at the 109th Annual Session General Assembly. Dr. Stuart has served as Councilor for the Sixth District from 1958 to 1961 and as Council Chairman for 1960-1961. We look forward to a good year with Dr. Stuart as President and urge support from all members of the Association.

Dr. Stuart was born in Calais, Maine, April 14, 1896, the son of Harry W. and Nancy G. Stuart. He was educated at Georgetown University, Washington, D.C., and received his medical degree from Georgetown Medical School in 1921. He practiced in Calais, Maine from 1921 to 1925; Sangerville, Maine from 1925 to 1929 and in Guilford, Maine since 1929.

He is a member of the American Medical Association, the Maine Medical Association and is a member and Past President of the Piscataquis County Medical Society.

Dr. and Mrs. Stuart, the former Ruth Groves of Milltown, Maine, reside at 27 High Street in Guilford.

Elected at the 109th Annual Session  
of the Maine Medical Association

Rockland, Maine

June 17, 18, 19, 1962

*President*

RALPH C. STUART, M.D., Guilford

*President-elect*

ERNEST W. STEIN, M.D., Pittsfield

*Councilor, Third District*

JOHN F. DOUGHERTY, M.D., Bath

*Councilor, Fourth District*

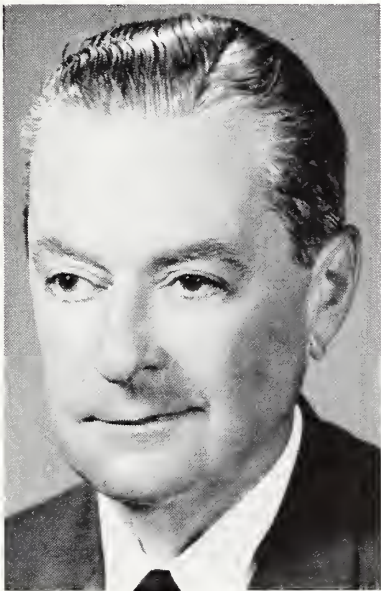
GEORGE E. SULLIVAN, M.D., Fairfield

*Council Chairman*

THOMAS A. MARTIN, M.D., Portland



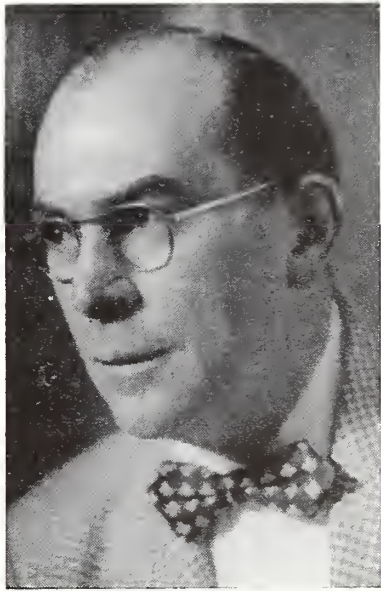
DR. STEIN



DR. DOUGHERTY



DR. SULLIVAN



DR. MARTIN

# Maine Medical Association

## STANDING COMMITTEES 1962-1963

Standing Committees for 1962-1963 as proposed by the Nominating Committee and approved at the Second Meeting of the House of Delegates of the Maine Medical Association at Rockland, Maine, June 17, 1962.

### Nominating Committee

- 1st District* — ROBINSON L. BIDWELL, M.D., Portland  
*2nd District* — PAUL J. B. FORTIER, M.D., Lewiston  
*3rd District* — MERRILL J. KING, JR., M.D., Rockland  
*4th District* — GEORGE E. SULLIVAN, M.D., Fairfield  
*5th District* — RUSSELL G. WILLIAMSON, M.D., Blue Hill  
*6th District* — LINUS J. STITHAM, M.D., Dover-Foxcroft — *Chairman*

### Scientific Committee

- James E. Poulin, M.D., 177 Main St., Waterville (1 yr.) —  
 Chairman  
 Robert L. Ohler, M.D., Veterans Administration, Togus (2  
 yrs.)  
 Albert L. Hunter, M.D., Knox County Hospital, Rockland  
 (3 yrs.)

### Legislative Committee

- Brinton T. Darlington, M.D., Westwood Rd., Augusta (2  
 yrs.) — Chairman  
 M. Tieche Shelton, M.D., 61 Winthrop St., Augusta (2  
 yrs.)  
 Charles A. Hannigan, M.D., 85 Goff St., Auburn (1 yr.)  
 John F. Andrews, M.D., 20 West St., Boothbay Harbor (1 yr.)  
 John D. Denison, M.D., 105 Brunswick Ave., Gardiner (3 yrs.)

### Medical Advisory Committee

- Thomas A. Martin, M.D., 157 Pine St., Portland — Chairman  
 Gerald H. Donahue, M.D., 4 Station St., Presque Isle  
 Philip L. Gray, M.D., Blue Hill  
 C. Harold Jameson, M.D., Medical Arts Bldg., Rockland  
 George L. Maltby, M.D., 31 Bramhall St., Portland  
 Clement S. Dwyer, M.D., 205 French St., Bangor  
 Allan J. Stinchfield, M.D., P.O. Box 343, Augusta.

### Board Of Ethics And Dicipline

- William F. Mahaney, M.D., 338 Main St., Saco (1 yr.) —  
 Chairman  
 Robinson L. Bidwell, M.D., 31 Bramhall St., Portland (1 yr.)  
 Eugene E. O'Donnell, M.D., 32 Deering St., Portland (2 yrs.)  
 Armand Alberr, M.D., 193 Main St., Van Buren (2 yrs.)  
 James A. MacDougall, M.D., 303 Penobscot St., Rumford (3  
 yrs.)  
 Ralph A. Goodwin, Sr., M.D., 56 Denison St., Auburn (3 yrs.)

### Rural Health Committee

- Isaac Nelson, M.D., Box 336, Greenville (2 yrs.) — Chairman  
 George W. Bostwick, M.D., Newcastle (2 yrs.)  
 Robert H. Pawle, M.D., 8 Walcott Ave., Falmouth (1 yr.)  
 Harland G. Turner, M.D., Box 38, Norridgewock (1 yr.)  
 Donald E. Allen, M.D., Sebago Lake (3 yrs.)  
 Alexander W. Magocsi, M.D., York (3 yrs.)

### Committee On Recruitment, Aid And Placement

- Paul H. Pfeiffer, M.D., 14 Gilman St., Waterville (1 yr.)  
 — Chairman  
 Ralph Zanca, M.D., 86 Pine St., Lewiston (1 yr.)  
 Charles W. Capron, M.D., 22 Bramhall St., Portland (2 yrs.)  
 Robert J. Barrett, Jr., M.D., Cor. Union & James Sts., Bangor  
 (2 yrs.)  
 Peter B. Aucoin, M.D., 87 Congress St., Rumford (3 yrs.)

### Public Relations Committee

- Donald F. Marshall, M.D., 142 High St., Portland (2 yrs.)  
 — Chairman  
 Earle M. Davis, M.D., 2 School St., Waterville (2 yrs.)  
 Warren C. Baldwin, M.D., 42 Deering St., Portland (1 yr.)  
 Robert J. Barrett, Jr., M.D., Cor. Union & James Sts., Bangor  
 (3 yrs.)  
 John A. Root, M.D., 22 White St., Rockland (3 yrs.)

### Health Insurance Committee

- Francis A. Winchenbach, M.D., 910 Washington St., Bath  
 (2 yrs.) — (Lincoln-Sagadahoc) — Chairman  
 Daniel R. Shields, M.D., 369 Main St., Lewiston (1 yr.) —  
 (Androscoggin)  
 Harry M. Helfrich, Jr., M.D., 122 Academy St., Presque Isle  
 (1 yr.) — (Aroostook)  
 Albert Aranson, M.D., 39 Deering St., Portland (2 yrs.) —  
 (Cumberland)  
 Paul A. Fichtner, M.D., 781 High St., Bath (3 yrs.) —  
 (Franklin)  
 Robert F. Russell, M.D., Penobscot (1 yr.) — (Hancock)  
 Kenneth W. Sewall, M.D., 2 School St., Waterville (1 yr.) —  
 (Kennebec)  
 Edward K. Morse, M.D., 22 White St., Rockland (1 yr.) —  
 (Knox)  
 Ake Akerberg, M.D., 10 Maple St., South Paris (3 yrs.) —  
 (Oxford)  
 Leonard G. Miragliuolo, M.D., 10 Maple St., Bangor (3 yrs.)  
 — (Penobscot)  
 Charles H. Lightbody, M.D., No. Main St., Guilford (2 yrs.)  
 — (Piscataquis)  
 Edgar J. Smith, M.D., 1 Park St., Fairfield (3 yrs.) — (Som-  
 erset)  
 George L. Temple, M.D., Fahey St., Belfast (2 yrs.) —  
 (Waldo)

James C. Bates, M.D., Eastport (3 yrs.) — (Washington)  
 Roger J. P. Robert, M.D., 331 Main St., Saco (2 yrs.) —  
 (York)

### Members of the Advisory Committee to the Health Insurance Committee

Maine Society of Anesthesiology — Clement S. Dwyer, M.D.,  
 205 French St., Bangor  
 Maine Chapter, American Academy of General Practice —  
 John D. Denison, M.D., 105 Brunswick Ave., Gardiner  
 Maine Society of Obstetrics and Gynecology — E. Allan  
 McLean, M.D., 29 Deering St., Portland  
 Maine Chapter, American Academy of Pediatrics — Everett  
 A. Orbeton, M.D., 131 Chadwick St., Portland  
 Maine Society of Internal Medicine (Includes Medical Specialty  
 Group) — Albert Aranson, M.D., 39 Deering St., Port-  
 land  
 Maine Eye Group — Nobody appointed  
 Maine Radiological Society — John F. Gibbons, M.D., 22  
 Bramhall St., Portland  
 Maine Chapter, American College of Surgeons — John F.  
 Reynolds, M.D., 216 Main St., Waterville  
 Maine Trauma Committee — John A. Woodcock, M.D., 35  
 Second St., Bangor

Ear, Nose and Throat Group — John E. Whitworth, M.D.,  
 116 Hammond St., Bangor

### Committee On Credentials

Robinson L. Bidwell, M.D., 31 Bramhall St., Portland (1 yr.)  
 — Chairman  
 James C. Bates, M.D., Eastport (1 yr.)  
 Charles R. Glassmire, M.D., 58 Deering St., Portland (2 yrs.)  
 Samuel Rideout, M.D., 3 Green St., Fort Fairfield (2 yrs.)  
 Thomas G. Harvey, M.D., 59 Mayo St., Caribou (3 yrs.)

### Delegate And Alternate To A.M.A. January 1, 1961 to January 1, 1963

Delegate — Asa C. Adams, M.D., 68 Main St., Orono  
 Alternate Delegate — George J. Robertson, M.D., 33 College  
 Ave., Waterville

### Delegate And Alternate To A.M.A. January 1, 1963 to January 1, 1965

Delegate — Asa C. Adams, M.D., 68 Main St., Orono  
 Alternate Delegate — Paul H. Pfeiffer, M.D., 14 Gilman St.,  
 Waterville

## SPECIAL COMMITTEES 1962-1963

Special Committees for 1962-1963 as appointed by the President, Ralph C. Stuart, M.D., of Guilford

### Committee On Alcoholism

Gilmore W. Soule, M.D., 22 White St., Rockland — Chairman  
 Paul A. Jones, M.D., Union  
 Jacob M. Jackler, M.D., 14 Gilman St., Waterville

### Veterans Affairs Committee

William C. Burrage, M.D., 57 Deering St., Portland —  
 Chairman  
 Robert L. Ohler, M.D., Veterans Administration, Togus  
 Lorrimer M. Schmidt, M.D., Veterans Administration, Togus

### Committee On Aging

Carleton H. Rand, M.D., 219 Oak St., Lewiston — Chairman  
 Eustache N. Giguere, M.D., 90 Webster St., Lewiston  
 Robert O. Kellogg, M.D., 316 State St., Bangor  
 Brinton T. Darlington, M.D., Westwood Rd., Augusta  
 Leon R. Jellerson, M.D., 34 Winter St., Sanford

### Arthritis Committee

Philip P. Thompson, Jr., M.D., 131 Chadwick St., Portland —  
 Chairman  
 Joseph A. Marshall, M.D., 177 Main St., Waterville  
 Philip L. Archambault, M.D., 346 Main St., Lewiston  
 Charles R. Glassmire, M.D., 58 Deering St., Portland

### Amy W. Pinkham Fund Committee

Norman H. Nickerson, M.D., Greenville — Chairman  
 Virginia C. Hamilton, M.D., 900 Washington St., Bath  
 Albert M. Carde, M.D., 33 Elm St., Milo  
 Thomas A. Foster, M.D., 131 State St., Portland  
 Ella Langer, M.D., State House, Augusta  
 Forrest B. Ames, M.D., 255 Hammond St., Bangor

### Committee On Mental Health

Guy N. Turcotte, M.D., 38 Deering St., Portland — Chairman  
 Francis H. Sleeper, M.D., Box 724, State Hospital, Augusta  
 Harold A. Pooler, M.D., State Hospital, Bangor  
 William E. Schumacher, M.D., 14 Westwood Rd., MD "B",  
 Augusta  
 Philip Blinder, M.D., 128 Broadway, Bangor

### Committee For Maternal And Child Welfare

Alice A. S. Whittier, M.D., 143 Neal St., Portland —  
 Chairman  
 Philip B. Chase, M.D., 36 Main St., Farmington  
 William M. Shubert, M.D., 317 State St., Bangor  
 Ella Langer, M.D., State House, Augusta  
 Benjamin L. Shapero, M.D., 142 Pine St., Bangor

(To be continued in August issue)

## Diseases Of The Heart And Blood Vessels

### Clinical Notes\*

Basal blood pressures measured by a defined technic help us to decide which hypertensive patients need treatment. . . . .

The basal blood pressure: — Under defined basal conditions with a sedative, a night's rest in hospital and repeated measurements of the blood pressure for 10 to 15 minutes in a single, quiet room, a pressure is obtained which appears to be a physiologic constant for the subject at the time. This we call the basal blood pressure. The labile fraction of the blood pressure — casual blood pressure minus basal blood pressure — we call the supplemental blood pressure. In an 8 year follow-up study of substantially untreated patients we found that the mortality was closely related to the basal blood pressure, but not correlated or even slightly negatively correlated with the supplemental blood pressure.

*Sir F. Horace Smirk, THE AMERICAN JOURNAL OF CARDIOLOGY, Vol. 9:90, January, 1962, Hypotensive Therapy in Essential Hypertension.*

It should be kept in mind that certain reports based on insurance statistics and on examinations of candidates for induction into the armed forces during World War II suggest that transient elevations of blood pressure may be of future clinical significance. — Ed.

Increased SGO-T values must be viewed with caution in patients with a combination of (a) suspected myocardial infarction, (b) normal or equivocal ECG, (c) biliary tract disease, and (d) prior narcotic administration.

*S. M. Mossberg, et al, ARCHIVES OF INTERNAL MEDICINE, Vol. 109: 429, April, 1962, Serum Enzyme Activities Following Morphine.*

The finding of a normal SGO-T value in the face of a history compatible with myocardial infarction should not be construed as excluding myocardial infarction. The time element and the number of SGO-T determinations may determine the frequency with which SGO-T is elevated. — Ed.

Ed. — JACOB B. DANA, M.D.

\*Submitted by the Maine Heart Association, Inc.



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

# Department of Health and Welfare

## Guidelines For The Use Of Oral Vaccine\*

Guidelines dealing specifically with the use of the oral vaccine are, in general, based upon the February, 1962 recommendations of Sub-committee One of the Advisory Committee on Poliomyelitis Control.

1. The program must have careful planning and achieve a maximum of support from official and voluntary health and medical groups. The active support of the organized medical professions is essential. Civic organizations — Parent Teachers Association, Association of Retail Druggists, National Foundation, Sister Kenny Foundation, various luncheon clubs, Federation of Women's Clubs, the communications media, and others — can offer material help.

2. The plans should assure the ready availability of the vaccine in all areas of the community and for all persons within the selected target groups. Special emphasis must be directed to those areas and population groups having the lowest levels of immunization. Community-wide programs should achieve the immunization of the maximum number of persons, but no less than 80% of the preschool children in all socioeconomic groups.

3. It is recommended that the three types of oral vaccines be administered sequentially, each in monovalent form at intervals of about six weeks. Special efforts will be necessary to maintain a high level of community interest and public response to obtain the necessary coverage.

4. Optimally, large scale immunization campaigns with oral poliovirus vaccines should be conducted during the winter or spring months. This consideration, however, should not preclude initiation of such programs in communities that have been unable to obtain sufficient vaccine sooner.

5. A continuing program of immunization of infants should be incorporated as an essential feature of all organized community-wide programs. Unless a high

proportion of infants is immunized during the first year of life, the benefit of a mass program will be short-lived. A continuing program of immunization of infants should be initiated as soon as possible in all communities and should continue indefinitely without regard to season. It can be started prior to and independent of mass community-wide programs.

*Epidemic Use.* Whenever epidemic poliomyelitis threatens an area, immediate mass use of type specific monovalent oral poliovaccine should be initiated. Adequate constant local surveillance and careful epidemiological appraisal must determine the extent of the epidemic threat and the population exposed. A limited reserve of oral poliovaccine for studies in epidemic use is maintained by the Communicable Disease Center. Allocations from this reserve may be made on request of State health officers upon presentation of evidence of the occurrence of at least three cases of poliomyelitis within one month, two caused by the same virus type. The community organization for adequate administration of the vaccine and the maintenance of effective surveillance must be assured.

*Dosage Schedules.* The following basic dosage schedules are recommended:

1. *Formaldehyde-Inactivated Vaccine.* Immunization should be initiated in infants between six weeks and three months of age according to the following schedule:

Number of Doses	Intervals from Previous Dose
First	—
Second	6 weeks
Third	6 weeks
Fourth	6 months or longer

The same schedule may be used for other age groups in the interest of achieving higher levels of immunity before the 1962 poliomyelitis season.

After the recommended four doses have been administered, additional doses (boosters) are specifically indicated for special reasons, such as the threat of an epidemic or travel to a hyper-endemic area.

(Continued on Page 180)

\*This is the second and last of two articles on the general subject of Poliomyelitis Prevention. The first, Poliomyelitis Prevention — 1962 was published in the June issue of The Journal.

# Unproven Cancer Therapy And The American Cancer Society\*

ROALD N. GRANT, M.D.\*\*

On June 18, 1731 Benjamin Franklin wrote to his sister, "I know cancer of the breast is often thought of to be incurable, yet we have here in town a kind of shell made of some wood cut at a proper time by some men of great skill which has done wonders in this disease among us, being worn for some time on the breast. I am not apt to be superstitiously fond of believing such things, but the instances are so well attested as to convince the most incredulous." Such naivete concerning cancer nostrums on the part of some of our citizens, has persisted up to the present day.

Less than a decade ago, the virus etiology of cancer theory was looked upon with considerable skepticism by the scientific community and the advocates were candidates for inclusion in the new or unproved cancer field. Yet today this is a respected opinion openly expressed by prominent physicians and scientists. The title chosen by the American Cancer Society, New or Unproved Methods of Cancer Treatment Committee, was formed in 1954 as the "Quackery Committee." However, the Committee took action to remove quackery from its title and adopted its present name because the word "quackery" implied pre-judgment. Also the new title places emphasis on the unproven method rather than the unorthodox individual. The Committee wisely recognized that concentrating attention on the unproven method rather than the individual is sound for several reasons. First, proponents of new and unproved methods of cancer therapy eventually either die or turn to other areas of interest, but unfortunately, the method lingers on. Also individuals tend to flourish under the spotlight of attention, while methods become less important as more becomes known about them. Moreover, individuals can take retributive action, justifiably or not, while methods cannot. Finally, concentrating on an individual often leads into questions of motivation which cannot be scientifically appraised while methods and materials can be carefully analyzed and evaluated.

In carrying out the American Cancer Society's objectives in this field, an Information Center has been developed for the collection and distribution of materials on new and unproved methods of cancer diagnosis and cure. This unique collection, which is one of the principal repositories for such information in this country, includes the voluminous file materials on this subject which were turned over to the American Cancer Society by the Committee on Cancer Diagnosis and Therapy of the National Research Council in 1957.

One of the fundamental concepts in controlling new and unproved methods of cancer therapy is to attack it at the local level because it is in the local community that the cancer victim and his family first come in contact with cancer nostrums in a manner which can be devastating. It is at this time that most lives are either lost or saved from cancer. Accordingly, the American Cancer Society has fostered the formation of State Cancer Commissions or Committees to assume responsibility for collecting and evaluating scientific and clinical evidence on various claims on the diagnosis and treatment of cancer. State control of unproven cancer therapy has also been fostered through the formulation of model legislation to assist states contemplating laws of this type. Such legislation to create a body having statutory standing with rights to investigate, hold hearings, and make reports of a privileged nature was first enacted in this country by the California Legislature in June 1959 and similar legislation has since been passed in Nevada, Colorado and Kentucky.

Information on the various unproven methods of cancer therapy has not been easily available because such material is not ordinarily published in medical journals. The American Cancer Society has recently taken action to fill the gap by utilizing the pages of its medical journal. — **CA — A Cancer Journal For Clinicians**. An important feature of each issue, which goes to over 125,000 physicians, is the statement on various new and unproved methods of cancer therapy prepared from the files of the National Office.

The Society's new and unproven methods of treatment program which supplies information to the public, writers, governmental agencies, medical societies and individual physicians is continuously growing. While it is neither a pleasant nor an easy road to follow, it is a most important one and we have no choice but to move forward with determination and skill in order that the thousands of new victims of cancer each year get only the best of America's finest medical care.

\*This is the first of a series of articles submitted by the Professional Education Committee of the Maine Cancer Society, Eugene E. O'Donnell, M.D., Portland, Maine, Chairman.

\*\*Director of Professional Education, American Cancer Society and Editor of **CA — A Cancer Journal for Clinicians**.

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## THE BASIC ACTION

ENOVID closely mimics the balanced progestational-estrogenic action of the functioning corpus luteum. This action is readily understood by a simple comparison. In effect, ENOVID *induces a physiologic state which simulates early pregnancy—except that there is no placenta or fetus.* Thus, as in pregnancy, the production or release of pituitary gonadotropin is inhibited and ovulation suspended; a pseudodecidual endometrium ("pseudo" because neither placenta nor fetus is present) is induced and maintained.

Further, during ENOVID therapy, certain symptoms typical of normal pregnancy may be noted in some patients, such as nausea—which is usually mild and disappears spontaneously within a few days—breast engorgement, some degree of fluid retention, and often a marked sense of well-being. There is no androgenicity. ENOVID *is as safe as the normal state of pregnancy.*

## THE BASIC APPLICATIONS

**1. Correction of menstrual dysfunction.** *Emergency* treatment of severe dysfunctional uterine bleeding is promptly effective following the administration of ENOVID in larger doses. *Cyclic* therapy with ENOVID controls less severe dysfunctional uterine bleeding. In amenorrhea *cyclic* therapy with ENOVID establishes a pseudodecidual endometrium providing the patient has endometrial tissue capable of response.

**2. Ovulation suppression (to suspend fertility).** For this purpose ENOVID is administered *cyclically*, beginning on day 5 through day 24 (20 daily doses). The ovary remains in a state of physiologic rest and there is no impairment of subsequent fertility. When ENOVID is prescribed for this *cyclic* use over prolonged periods, a total of twenty-four months should not be exceeded until continuing studies indicate that its present lack of undesired actions continues for even longer intervals. Such studies are now in their seventh year and will regularly be reviewed for extension of the present recommendation.



*...unfettered*

**3. Adjustment of the menses for reasons of health** (impending hospitalization for surgery, during treatment of Bartholin's gland cysts, acute urethritis, rectal abscess, trichomonal or monilial vaginitis), or other special circumstances considered valid in the opinion of the physician. For this purpose ENOVID may be started at any time in the cycle up to one week before expected menstruation. Upon discontinuation, normal cyclic bleeding occurs in three to five days.

**4. Endometriosis.** *Continuous* therapy with ENOVID corrects endometriosis by producing a pseudodecidual reaction with subsequent absorption of aberrant endometrial tissue.

**5. Threatened and habitual abortion.** ENOVID should be used as *emergency* treatment in *threatened abortion* although symptoms may occur too late to be reversible. *Continuous* therapy with ENOVID in *habitual abortion* is based on the physiology of pregnancy. ENOVID provides balanced hormone support of the endometrium, permitting continuation of pregnancy when endogenous support is otherwise inadequate.

**6. Endocrine infertility.** ENOVID has been used successfully in *cyclic* therapy of endocrine infertility, promoting subsequent pregnancy through a probable "rebound" phenomenon.

## THE BASIC DOSAGE

Basic dosage of ENOVID is 5 mg. daily in *cyclic* therapy, beginning on day 5 through day 24 (20 daily doses). Higher doses may be used with complete safety to prevent or control occasional "spotting" or breakthrough bleeding during ENOVID therapy, or for rapid effect in the *emergency* treatment of dysfunctional uterine bleeding and threatened abortion.

ENOVID is available in tablets of 5 mg. and 10 mg. Literature and references, covering more than six years of intensive clinical study, available on request.

SEARLE

*Research in the Service of Medicine*

DEPARTMENT OF HEALTH AND WELFARE — Continued from Page 177

2. Oral Vaccine (monovalent).

(a) Infants: Immunization should be initiated between six weeks and three months of age and subsequent doses given according to the following schedule:

Number of Doses	Type	Intervals from Previous Dose
First	I	_____
Second	III	6 weeks
Third	II	6 weeks
Fourth	I, II, & III	6 months or longer
All Others (including community use)		

Number of Doses	Type	Intervals from Previous Dose
First	I	_____
Second	III	6 weeks
Third	II	6 weeks

For organized community use during the poliomyelitis season of 1962, the availability of vaccine may necessitate changes in the above sequence of types. The interval between doses may be as short as 4 weeks if vaccination is begun in the spring or summer, or longer than six weeks if necessitated by shortage of specific type of vaccine or other delays.

# News, Notes and Announcements

**Department Of Health And Welfare  
Division Of Maternal And Child Health  
Including Services For Crippled Children  
(By Appointment Only)**

## Orthopedic Clinics

Augusta — Augusta General Hospital  
1:00 p.m.: Aug. 23  
Bangor — Eastern Maine General Hospital  
1:00 p.m.: July 26, Sept. 27  
(Several will be two-session clinics)  
Fort Kent — Peoples Benevolent Hospital  
10:00 a.m.: Sept. 12  
Lewiston — Central Maine General Hospital  
9:00 a.m.: July 20, Aug. 17, Sept. 21  
Portland — Maine Medical Center  
9:00 a.m.: Aug. 13, Sept. 10  
Presque Isle — Arthur R. Gould Memorial Hospital  
9:00 a.m. and 12:30 p.m.: Sept. 11  
Rockland — Knox County Hospital  
1:30 p.m.: Aug. 16  
Rumford — Community Hospital  
1:30 p.m.: Sept. 19

## Cardiac Clinics

Bangor — Eastern Maine General Hospital  
9:00 a.m.: July 27, Aug. 10, 24, Sept. 14, 28  
Portland — Maine Medical Center  
9:00 a.m.: Every Friday (holidays excepted)

## Cleft Palate Evaluation Committee

Portland — Maine Medical Center  
10:00 a.m.: Aug 14

## Pediatric Clinics

Bangor — Eastern Maine General Hospital  
1:30 p.m.: July 27, Aug. 24, Sept. 28  
Fort Kent — Peoples Benevolent Hospital  
10:00 a.m.: July 25  
Presque Isle — Arthur R. Gould Memorial Hospital  
1:30 p.m.: Sept. 26  
Waterville — Thayer Hospital  
1:30 p.m.: Aug. 7, Sept. 4

## Clinics For Mentally Retarded Preschool Children

Waterville — Thayer Hospital  
9:00 a.m.: July 18, Aug. 1, 15, 29, Sept. 5, 19

## Adolescent Clinics

Portland — Maine Medical Center  
1:00 p.m.: July 25, Aug. 22, Sept. 26

## Cystic Fibrosis Clinics

(In conjunction with the Maine Medical Center, Portland)  
Portland — Maine Medical Center  
9:00 a.m.: July 17, 18, Aug. 21, Sept. 18, 19

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# The Journal of the Maine Medical Association

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Brunswick, Maine, August, 1962

No. 8

## Lymphangiography

CHRIS A. LUTES, M.D.\* AND DONALD F. MARSHALL, M.D.\*\*

### INTRODUCTION

During the past few years there has been renewed interest in lymphangiography, the radiologic demonstration of lymph vessels and nodes. Most of this new work is based on the clinical technique and results of Kinmonth.<sup>1,6</sup> However, the first roentgenologic demonstration of lymph nodes was reported by Menville<sup>7</sup> who succeeded in showing the popliteal, inguinal, axillary, and mesenteric lymph nodes by injecting thorium dioxide in the tails and legs of dogs and rats. Pfahler,<sup>8</sup> by accident, in 1932, was first to radiographically demonstrate lymphatics in man. In the course of studying the sinuses by placing an iodized poppy seed oil in the antra, he noted late filling of the lymphatics of the head and neck.

Subsequently, numerous publications have appeared which until recent years were mostly in the European literature. However, the greatest impetus in this field was given by Kinmonth<sup>1,6</sup> with his extensive investigation of lower limb lymphatics. This basic technique of intralymphatic injection of radiopaque dye has since been used to study lymph drainage from the penis, scrotum, breast, arm, and leg, in children and in adults.<sup>9,17</sup>

The purpose of this paper is to review briefly lymphangiography and to present examples from the fourteen cases which have been studied at the Maine Medical Center.

### TECHNIQUE

The technique used is basically that described by Wallace<sup>18</sup> with but slight modification. The intralymphatic injection of Ethiodol® (a radiopaque ethylester of poppy seed oil containing 37% iodine) is accomplished in the following manner. Two to three tenths of a cc of 0.5% Evans blue dye is injected intradermally into the web space of the first and second toe after first anesthetizing the area with 1% procaine. A vertical cut down is then made 2-3 cm proximal to this site revealing one or more lymphatic channels which have selectively absorbed the Evans blue dye. The appearance of the dye can be facilitated by a gentle milking motion from the point of dye injection to the cut down site. After some experience the lymphatics can sometimes be identified even though they contain no dye. Using a #25 needle minus the hub and tightly adapted to a fine polyethylene catheter, the lymph duct is then cannulated. This is the most trying part of the procedure and sometimes it is time consuming. Proper cannulation is assured by the disappearance of proximal dye and or distention of the lymphatic when saline or procaine is infused. Depending on the size of the lymphatic a #5-0 silk tie is sometimes placed about it and the needle to prevent back flow leak of dye. Using an improvised screw type manual feeder which holds a 10 cc syringe, from 7 to 10 cc of Ethiodol is slowly injected, although for visualizing just the inguinal and pelvic nodes as little as 3 cc has sufficed. To prevent rupture of the lymphatic and leak of dye, the Ethiodol is infused slowly, taking about 45-60 minutes for 10 cc. The entire procedure minus x-ray time takes an average of two

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FIG. 1. Demonstration of lymph nodes in mediastinum and supraclavicular areas. Normal.

FIG. 2. Inguinal, pelvic, and para-aortic lymph nodes interpreted as normal.

FIG. 3. Abnormal matting of lymph nodes and dilatation and tortuosity of lymphatics due to metastatic carcinoma of the cervix.



FIG. 1

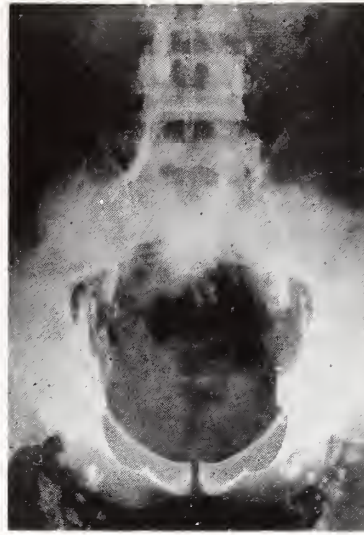


FIG. 2

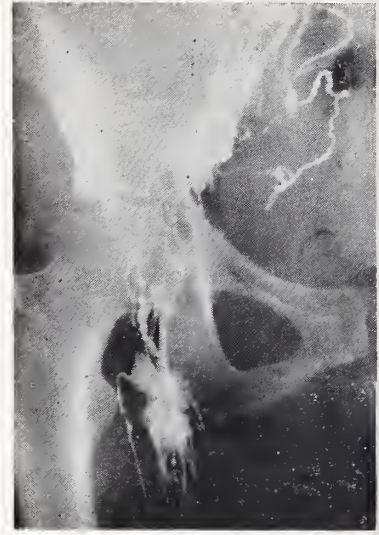


FIG. 3

hours. During the infusion the patient sometimes experiences a warm or very mild burning sensation along the course of the lymphatics. After the infusion is completed the wound edges are approximated with #5-0 silk sutures. X-rays taken immediately after completion of injection demonstrate the lymph channels of the extremity and the regional nodes. Best demonstration of node architecture is obtained about twenty four hours after injection. Dye remains in the nodes for as long as three months.

Direct sky blue 4%<sup>13,14</sup> or patent blue v can also be used for identifying lymphatics. Numerous radio-paque dyes have been tried including angiopac (an aqueous suspension of ethyl-iodostearate), lipiodol, thorotrast,<sup>12</sup> umbradil,<sup>19</sup> hypaque,<sup>20</sup> miokon,<sup>21</sup> iodipin, 70% diodone,<sup>16</sup> triopac 400 (similar to diodrost) and joduran<sup>10</sup> (diodrost preparation with hyaluronidase), but none have proven as satisfactory as Ethiodol.

#### COMPLICATIONS

Complications to date have been few and minimal, consisting of mild burning sensation along the lymphatics during injection and temporary blue discoloration of the foot at the site of injection of the identification dye. Other potential and theoretical complications include the following: hypersensitivity reaction to the dye; wound infection; lymphangitis; thrombophlebitis; lymphatic obstruction and oil embolism.<sup>13,15</sup> Oil embolism (due to the inadvertent injection of dye into small veins mistaken for lymphatics) has been reported. In one case non fatal pulmonary infarction was produced when a large volume (35cc) of contrast material was introduced.<sup>14</sup>

#### RESULTS

Using this technique it has been possible to study the lymphatics of both upper and lower extremities, penis, scrotum, and the regional nodes including inguinal, pelvic, para-aortic, supraclavicular, epitrochlear, and axillary lymph nodes and the thoracic duct.

To date our studies have been confined to the lower

extremities in fourteen patients with cancer involving the ovary, bladder, cervix, prostate, and testicle, and one patient with primary tuberculous peritonitis.

Figures 1 and 2 demonstrate normal lymph nodes from the femoral to the supraclavicular area in a young man with seminoma of the testicle. This helped confirm the clinical impression that no gross disease extended beyond the testicle, and aided the radiotherapist in correctly placing portals for therapy.

Different patterns of abnormal lymph nodes due to metastatic carcinoma are shown in figures 3, 4, 5, and 6. The nodularity of the dilated lymphatics in figure 3, appearing like chain sausages, is normal and due to the lymphatic valves.

The patient in figures 6 and 7 was suffering from carcinoma of the cervix stage III with a non functioning left kidney and hydronephrotic right kidney. Lymphangiography demonstrated abnormal pelvic lymph nodes attributed to metastatic disease, before radium and x-ray therapy (Fig. 6) and after therapy (Fig. 7.) There is visual evidence of primary and metastatic tumor response to therapy, both in the shrinkage of the lymph nodes and disappearance of the hydronephrosis.

#### DISCUSSION AND CONCLUSIONS

Lymphangiography is a useful tool in studying the anatomy and physiology of the lymphatic system in disease and health. The extensive research on the lower extremities has revealed some of the underlying pathology of primary lymphedema<sup>1-6</sup> and secondary changes in stasis ulcers<sup>21</sup> and in filarial lymphedema.<sup>16</sup> Study of normal lymphatic structures<sup>10,11,17,19</sup> has accurately delineated the fine lymphatics of the extremities disproving previously held misconceptions of lymphatic anatomy.<sup>23</sup> Interestingly under fluoroscopy direct communications between lymphatics and veins have been detected, in particular between the axillary nodes and subclavian vein, perhaps helping to explain the poor prognosis of carcinoma of the breast with axillary metastases.<sup>24</sup>

Other applications of this procedure are: 1. The



FIG. 4



FIG. 5

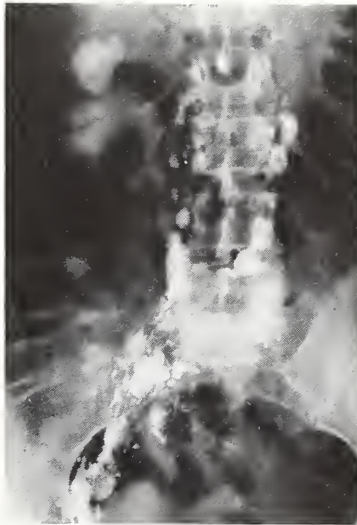


FIG. 6

FIG. 4. Pattern of lymph nodes partially replaced by metastatic carcinoma of the cervix. (Proven by biopsy) Residual barium in GI tract).

FIG. 5. Abnormal lymph nodes attributed to metastatic stage IV carcinoma of the cervix.

FIG. 6. Abnormal matting of lymph nodes. Stage III carcinoma of the cervix with right hydronephrosis.

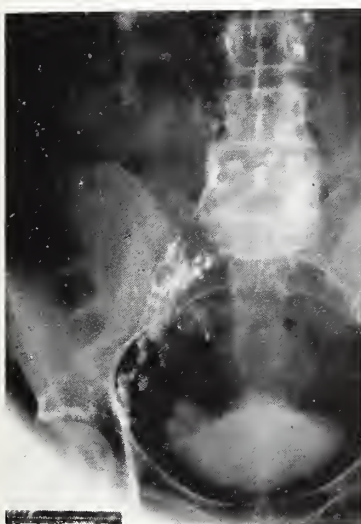


FIG. 7

FIG. 7. Same patient, as in Fig. 6, following radium and X-ray therapy, demonstrating shrinkage of nodes and presumably local tumor as evidenced by disappearance of hydronephrosis.

therapy. Sherman<sup>27</sup> in 1951, using a radioactive colloidal gold solution demonstrated the effect of intracervical and parametrial injection on regional lymph nodes which had selectively taken up the radioactive particles. Little if any investigation has been done with the intralymphatic injection of the non vesicant chemotherapeutic agents now available. This combined with surgery, in properly selected lesions of the extremity would seem a particularly useful research tool in cancer chemotherapy.

#### SUMMARY

A brief review of the history of lymphangiography has been made and a description of the technique, complications, and research application has been presented. Suggestions for the adaptability of this procedure to cancer therapy are proposed. Selected lymphangiograms from fourteen patients studied at the Maine Medical Center are presented.

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*Continued on Page 194*

assessing of tumor response particularly lymphoma to x-ray and chemotherapy;<sup>18,25</sup> 2. The demonstration of the etiology of retroperitoneal masses; 3. The determination of the need for extensive surgery; 4. The evaluation of completeness of removal of lymphatics after surgery;<sup>14</sup> 5. The accurate placing of portals for radiotherapy.

While the interpretation of anatomical changes in the fine lymphatic channels does not present much of a problem the reverse is true for lymph nodes. Certain disease entities present a fairly typical picture (eg. lymphoma)<sup>18,26</sup> which is not difficult to interpret. Other lesions present variable pictures. Of particular importance is the range of variation of pattern in normal lymph nodes, as pointed out by Fisher,<sup>9</sup> making difficult the selection of the normal from the abnormal. This variation is perhaps the greatest hindrance to this procedure becoming a diagnostic tool in assessing metastatic carcinoma. It may be resolved as more lymphangiograms are studied.

The techniques described above can be applied to cancer chemotherapy. As early as 1932 Menville<sup>7</sup> using radioactive thorium dioxide in studying lymph nodes suggested this to enhance the efficacy of radiation

# Renovascular Hypertension – Report Of A Case

FERRIS S. RAY, M.D.\*

Renovascular lesions as a cause of hypertension are being recognized with increasing frequency. The etiologic relationship between hypertension and these lesions via the "Goldblatt Mechanism"<sup>1,2</sup> is now fairly well established and with the continued improvement in the techniques of reconstructive vascular surgery more and more of these cases are being successfully treated.

We plan to present a case of what appeared to be congenital renal artery stenosis producing severe hypertension in a young girl which was treated successfully by an aorto-renal bypass graft. Some of the current thoughts regarding the diagnosis and treatment of renovascular hypertension will also be discussed.

## CASE REPORT

A girl, age 15, was admitted to the Maine Medical Center on January 25, 1962, for investigation of severe hypertension. She had complained of throbbing headaches for about four years, and in recent months they had become more constant and severe. She had also noted some recent blurring of vision associated with her headaches. There was no history of congestive heart failure or other abnormal cardio-respiratory symptoms. She was said to have had rheumatic fever at the age of 7 but this was not substantiated. Remainder of the past history and system review was essentially negative. The patient had two sisters who died of congenital heart disease and one brother living who has congenital heart disease. There was no family history of hypertension.

Physical examination revealed B.P. 185/135 (rt. arm) and 185/125 (lt. arm). Funduscopic examination showed grade I-II hypertensive changes with mild A-V nicking, copper-wire arterioles and an A/V ratio of 1/3. Examination of the heart revealed N.S.R. with a grade II soft systolic murmur. Abdominal examination was negative with no bruits heard.

Hemoglobin, WBC and urinalysis were normal, B.U.N. 15 mg. %, F.B.S. 80 mg. %, and urine culture revealed no growth. Serum electrolytes were all normal. Regitine test was negative. The following laboratory studies performed at the referring hospital (Central Maine General, Lewiston, Maine) are all within normal limits: creatinine 1.2 mg. %, P.S.P. excretion 45% in 15 minutes, cholesterol 139 mg., serology negative, sed. rate 9, urinary 17 ketosteroids 3.9 mg., 17 hydrocorticoids 3.6 mg., catecholamines 55 mcg. %. Urinary NA and K were normal. E.K.G. and E.E.G. were both normal.

I.V.P. showed the right kidney to be 1 cm. smaller than the left and showed a more dense concentration of dye in the right kidney than in the left. On January 30, 1962, a retrograde femoral renal angiogram was performed under local anesthesia† (FIG. 1). This study revealed a stenosis of the proximal portion of the right renal artery with post-stenotic dilatation. The stenotic area measured approximately 1.5 cm. in length and 2 mm. in diameter. The left renal arteries appeared normal. The patient experienced no complications from the arteriogram. On this first hospital admission

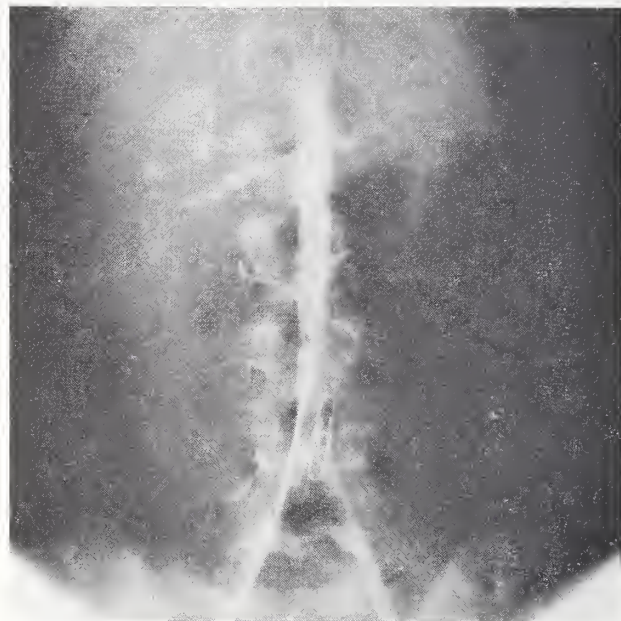


FIG. 1. Retrograde femoral renal angiogram revealing stenosis of right renal artery with post-stenotic dilatation.

blood pressure recordings ranged from 140/90 to 188/125.

On February 26, 1962, an 8 mm. dacron aorto-renal bypass was performed through a midline abdominal incision. Marked stenosis of the proximal 2 cm. of the right renal artery with post-stenotic dilatation was found. This had the gross appearance of a congenital lesion. The pressure gradient across the stenosis before bypass was 80 mm. Hg. After bypass the pressure gradient was reduced to 20 mm. Hg. The circulation to the right kidney was interrupted for 20 minutes while the anastomosis was constructed. The postoperative course was uneventful. The blood pressure remained elevated during the night of surgery but thereafter fell to normal levels and remained normal throughout the hospital course. The patient's headaches improved, and examination of her fundi prior to discharge revealed definite improvement in the angiospastic changes noted preoperatively with no A-V nicking and improvement in the A/V ratio to 1/2. The patient was last seen on April 9, 1962, at which time her blood pressure was 140/80, the fundi appeared normal and she had remained free of symptoms.

A postoperative retrograde femoral aortogram (Fig 2) performed four months after surgery revealed the aorto-renal bypass graft to be functioning well.

## DISCUSSION

It is impossible to establish rigid criteria which will allow one to suspect or make a clinical diagnosis of renovascular hypertension. It seems worthwhile, however, to outline certain clinical features which may suggest the possibility of renovascular pathology so that the few cases with correctable lesions will not be overlooked and so that unnecessary diagnostic tests and procedures will be avoided in others.

\*Attending surgeon, Maine Medical Center, Portland, Maine

†Performed by Dr. Niles L. Perkins, Jr., senior medical resident



FIG. 2. Postoperative retrograde femoral aortogram showing functioning aorto-renal dacron bypass graft.

The following clinical features strongly suggest the possibility of renal ischemia as the basis of hypertension.<sup>3</sup>

1. Young patients, usually under 35, with severe persistent hypertension without a familial history and with no other apparent cause.
2. Malignant hypertension of recent origin in older patients.
3. Hypertension which develops or worsens after renal infarction.

There is no set clinical pattern and the medical course of these patients cannot be predicated. The improvement or lack of improvements to anti-hypertensive drugs or bed rest has not been consistent and is generally of no value in differentiating renovascular from essential hypertension.<sup>4</sup> Physical examination reveals no specific diagnostic findings. Angiospastic eyeground changes may or may not be present. The presence of a soft systolic bruit heard best over the flanks or over the renal artery anteriorly is very suggestive of renal artery stenosis and should be sought for in all hypertensive patients. Routine laboratory test including the usual renal function tests does not aid in the diagnosis. One of the most helpful examinations which may suggest the presence of renovascular lesions, and which is said to be positive in about 75% of cases, is the routine intravenous pyelogram.<sup>5</sup> Although not pathognomonic the following findings on I.V.P. strongly suggest a functional stenosis of the renal vasculature:

1. Difference in length of the kidney of 1 cm. or more.
2. Delayed excretion of dye in one kidney compared with the other — suggesting impaired function (often paradoxical hyperconcentration may be seen in the abnormal kidney). This is due to production of less but more concentrated urine by the affected kidney.
3. Smaller kidney with smooth contours and narrow compact calyces.
4. Non-functioning kidney which is normal on retrograde pyelogram.

The intravenous pyelogram is a very important examination in hypertensive patients and should be examined carefully for the findings listed above.

Radioactive renograms are used as an aid in the diagnosis of renovascular lesions revealing delayed uptake in the kidney with reduced blood flow. Although helpful only in the presence of unilateral lesions, this procedure is used by some clinics as a method of screening hypertensive patients for further diagnostic test.<sup>6</sup> Split renal function tests<sup>7,8</sup> have been utilized in the diagnosis of renal hypertension but is not a definitive test and in most clinics is not used routinely. When performed carefully it is an important aid in cases where the angiogram may be doubtful or difficult to interpret.

The only definitive procedure to diagnose unilateral or bilateral renovascular pathology is renal angiography either by translumbar aortography or by retrograde femoral catheterization. Although each method of visualization of the renal artery has its proponents, both methods have proven reliable and safe when executed properly by experienced personnel. The retrograde femoral method is probably best suited for younger patients and the translumbar technique best for older patients who often have associated aorto-iliac occlusive disease.

The pathology of renal artery disease responsible for hypertension has not yet been adequately studied or clearly classified. Certain pathological lesions, namely the "fibromuscular hyperplasia" group, are not completely understood. The following is a revised list of the more commonly encountered lesions.<sup>9</sup>

#### *I Congenital*

- a. Stenosis of renal artery.
- b. "Fibromuscular hyperplasia."
- c. Renal artery aneurysm.
- d. Arterio-venous malformation (fistula, etc.).

#### *II Acquired*

- a. Atherosclerosis of renal artery.
- b. Thrombosis or embolus of renal artery.
- c. Trauma with thrombosis, A-V fistula, hematoma.
- d. "Vasculitis" of renal artery.
- e. Tumor (extrinsic or intrinsic).

Atherosclerotic plaques and congenital stenosis appear to be the most common lesions. "Fibromuscular hyperplasia" (usually found in patients under 40, mostly women), renal artery thrombosis and renal artery aneurysm are next in frequency. The presence of bilateral lesions is reported between 25%<sup>3</sup> and 35%<sup>10</sup> by various investigators.

The selection of these cases for surgical treatment is not simple and depends on a complete evaluation and individualized approach to each patient. The usual techniques of reconstructive vascular surgery are applied to revascularize the kidney as quickly and safely as possible. Some clinics employ hypothermia as an adjunct allowing more time to accomplish the recon-

struction,<sup>11</sup> but as a rule the renal artery can safely be clamped for 30 minutes without kidney damage. When dealing with renal artery stenosis involving the left kidney, the splenic artery has been utilized successfully to perform a spleno-renal shunt.<sup>12</sup> The spleen need not be removed as it will survive on the circulation from the short gastric vessels.

In general the best results have been obtained in cases similar to the one presented, i.e.: well marked stenosis with post-stenotic dilatation and a significant pressure gradient across the area of stenosis. It has also recently been suggested that an early fall in diastolic pressure, within 3 days after surgery, is a good indication of a successful result and promises a favorable prognosis.<sup>13</sup>

#### SUMMARY

A case of renal artery stenosis producing hypertension treated successfully by an aorto-renal bypass graft has been presented.

Some of the clinical features suggesting the possibility of renovascular disease as the cause of hypertension have been outlined and discussed.

The intravenous pyelogram and radioactive renogram are probably the two best examinations to be used for preliminary investigation of screening of patients for unilateral renovascular lesions.

Accurate renal angiograms, either by translumbar aortography or retrograde femoral catheterization, are necessary for a definitive diagnosis.

The best results to date have been observed in patients with well-defined stenosis, post-stenotic dilatation, significant pressure gradients and the lack of wide-spread arteriosclerotic disease.

With the careful selection of patients and the application of the proper surgical techniques, a high percent-

age of successful results in the treatment of renovascular hypertension should be realized.

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131 Chadwick Street, Portland, Maine



# Medical Audit At The Maine Medical Center

## Method And Evaluation

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The problem of accomplishing a satisfactory method of auditing the medical records of a large community hospital was presented to a group of six members of the medical staff at the Maine Medical Center. No practical or efficient method of auditing medical records had been evolved to our knowledge. The methods and results of this attempt will be described.

The specific goal of improved medical care to the patient is the hope of any such self-evaluation and critique. The various approaches to the problem were explored and will be outlined. The guides for such a survey were set. The highlights of our experiences of the year's work will be presented.

### Guides for a Medical Audit:

1. It was felt that such an audit should be of educational value to the entire staff rather than a censorship or discipline of a few individuals.

2. It should be kept within the practical range of the evaluating committee and the record room staff.

3. It should have limited specific goals which could be presented at regular intervals and be meaningful in themselves.

4. The limited short term surveys should be able to be coordinated with a long range survey of Medical Care in the hospital.

5. It should be known to the staff their colleagues are to peruse and evaluate the medical practice of each member. It is hoped that this in itself may improve the quality of the record and management of the patient.

### Possible Methods of Evaluation

A. Broad scrutiny of total medical care in the following categories:

1. Death causes.
2. Autopsied cases.
3. Specific diseases.
4. All hospital stays longer than 10 days.
5. Cost of hospitalization.
6. Diagnostic admissions.
7. Out Patient Services (medical).
8. Emergency Division Care (medical).
9. Special Care Unit (medical).
10. Deaths within 48 hours.

B. Narrow scrutiny of individual records for quality of medical care in the following categories.

1. Individual physician.
2. Special groups:

- a. Medical attending staff.
- b. General Practice Department.
- c. Courtesy Staff.
3. Specific Disease diagnoses.
4. Diagnostic Efficiency.
5. Therapeutic Efficiency.
6. Cost of Hospitalization.
7. Utilization and abuse of third party payers:
  - a. Voluntary health insurance.
  - b. Workmen's Compensation.
  - c. State aid cases
  - d. City Welfare cases.
  - e. Veterans
8. Service versus private care:
  - a. cost.
  - b. quality.
9. Medical consultations and consultations for medical cases.

It was decided that a simultaneous horizontal and vertical perspective would best accomplish the aims of our audit. By doing this evaluation comparative medical management would be facilitated.

The record room personnel were asked to list all records admitted during a year with the Discharge Diagnosis of the disease under study. This list included the Disease, Age, Sex, Service or Private, Length of Stay, the fact that the patient died or not, and if an autopsy was done. From this list were selected at random sample records to be carefully reviewed by the group. From 30 to 50 records from this list were selected so that each member of the group could carefully review 5 or 8 records at a session. This required about an hour. Specific points of care, errors of diagnosis or management, and omissions in the record itself were noted by each individual auditor. These points were then discussed by the entire group. A summary of the mutual errors and specific items of universal educational value were made and presented to the entire medical staff or house staff. The discussion period of the group generally required another hour so the total sessions required about two hours of the groups time.

### Highlights of the results of a year's Auditing.

#### Diabetes Mellitus:

Admissions:

364 Male 133 Female 229 % of total 3%

Deaths:

41 15 26

1. The management of diabetes by the intern resident staff was haphazard and frequently resulted in poor control of the diabetic state. This was particularly true if the diabetic patient was the primary responsibility of a non-medical service.

2. No patient died of uncontrolled diabetes.

3. The management of patients in diabetic coma should be under the direct supervision of the visiting staff and bedside attention by the house staff until the patient is out of coma.

4. The use of oral anti diabetic agents alone is to be condemned in the presence of infection, or in most cases for in-hospital management of diabetic complication.

5. The diagnosis "Diabetes Mellitus" as a primary diagnosis is used too loosely. It should be qualified for example, "Diabetic Acidosis" with or without coma. "Diabetic Mellitus" controlled or uncontrolled, or should be listed in its proper place as a secondary diagnosis if a specific disease or complication of the diabetic state caused the patient to be admitted to the hospital.

#### *Acute Gastroenteritis*

Admissions:	97	30	15 years and under.
		22	60 years and over.

Deaths 0

1. In almost all children the classical syndrome of nausea, vomiting and diarrhea were present. Hospitalization was specifically for the correction of or prevention of dehydration. No enteric pathogen was found or ever looked for, prompt recovery and discharge was the usual course.

2. The diagnosis among adults particularly the 20 to 60 year age group was made on tenuous grounds. It covered a multitude of conditions ranging from gastritis of acute and chronic alcoholism, gastritis medicamentosa to psychogenic gastrointestinal disorders. Diarrhea in this group was relatively infrequent as were cultures of the stool.

3. The disease in patients over sixty was generally a "waste-basket" diagnoses when no cause of nausea and vomiting was found. Carcinoma was searched for and not found. Many patients had pyuria without fever and B.U.N.'s were frequently not checked.

#### *Myocardial Infarction — Recent*

Admissions:	251	% of total	2%
Deaths	61		

1. Average length of stay; 24 days (excluding deaths).

2. Anticoagulants were used uniformly.

3. No death was attributable to over dosage of anticoagulants although a death was simultaneous with an excessively prolonged prothrombin time.

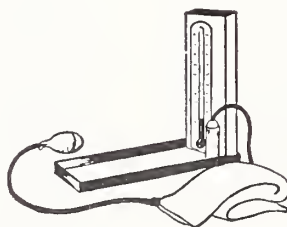
4. Diagnostic measures necessitating the moving of the patient should be delayed until myocardial infarction is excluded.

5. The use of drugs such as aminophyllin, large doses of nitroglycerin, and anti hypertensive drugs should not be administered in the face of frank or impending infarction.

#### CONCLUSION

An audit of all medical admissions of a large community hospital is not practical or possible. The various ways a selective audit could be accomplished were explored. A study of "specific diseases" in succession was found to be a practical method of evaluating a cross section of patient care and long section of individual physician's management of his patients. Some of the conclusions drawn from the application of this method of an audit are presented.

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# Mammography

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The awareness of radiography as a supplemental method of examining the breast has been slow in developing as compared with many other quickly accepted radiographic procedures. The results of x-ray examination of the breast were reported as early as 1930 by S. L. Warren.<sup>1</sup> Studies at this time were seriously hampered by lack of adequate film to record the short range of contrast found in soft tissue. The use of contrast media in mammography was reported by Romano and McFetridge in 1938.<sup>2</sup> This method of study, however, was unsuccessful, not only because of discomfort and the complications of retrograde injection of opaque material intraductally but also because inconclusive results were obtained in attempting to differentiate benign from malignant disease. In more recent years, the most persistent interest in this subject has been demonstrated by the prolific writings of J. Gershon-Cohen, H. Ingleby, and various associates from the Einstein Medical Center in Philadelphia.<sup>3</sup> Their interest began as early as 1937 and continues until the present. Their writings have contributed a great deal to the radiographic distinction of different histological densities and also to the development of improved x-ray techniques which overcome the technical problems of portraying soft tissue densities of such short contrast scale. Through his study of over one thousand cases, correlating the mammograms with the pathological findings, he was one of the first to establish definite radiographic criteria for differentiating malignant from benign disease and subdividing these into their various types.

However, the greatest impetus to the generalized acceptance of mammography was the report of Robert L. Egan in 1960.<sup>4</sup>

He reported over one thousand mammograms with 99% accuracy in the differentiation of benign from malignant disease. He also found nineteen clinically unsuspected carcinomas that were later confirmed by biopsy. Dr. Egan added refinements to film techniques and helped to establish further the radiographic criteria necessary for diagnosis. This high diagnostic accuracy coupled with the routine nature of these studies, requiring no special x-ray equipment, has been a strong stimulus to the development of mammography. At present, Dr. Egan is collecting material from radiologists all over the country for further evaluation of mammographic studies.

One reason for the slow advance of mammography has been the reluctance of clinicians to accept the

radiologist in the role of the predictor of histology. And yet the radiologist accepts this role in the prediction of the type of gastric ulcer or the type of gastric malignancy or in the predicted histology of renal tumors, bone tumors, and brain tumors. It should be stressed that this study in no way is intended to replace biopsy of the breast. In fact, it will probably result in a greater number of biopsies.

The success of this study rests largely on obtaining technically satisfactory films that will permit differentiation of the various soft tissue components of the breast. The technique used at the Maine Medical Center has evolved from trial and error, starting with the experience of other radiology departments. The choice of film is of prime importance. Our original efforts were with regular Kodak film. Later, Kodak Industrial Film, Type M, was used with much improved contrast.

Our own innovation has been the use of Ansco Superay "A-B" Sandwich Pak Industrial Film. This consists of a pack of two industrial films of different speeds and results in two films of different density with one exposure. The lighter film permits study of the skin, the nipple, and the anterior thinner portion of the breast. The darker film shows better penetration of the thicker posterior breast tissue and chest wall. This type of film pack greatly reduces the need for repeat films and more x-ray exposure. This fine grain industrial film provides good detail over a short contrast range.

The technique factors are 23-32 KVP with exposures of 100-300 milliamperere seconds at an average film-focal distance of 24 inches. All added filter is removed from the machine, and the field must be coned as much as the size of the breast will permit.

Routinely, two views of each breast are obtained. The first is a cranio-caudal view with the film under the breast and the x-ray cone perpendicular to it against the chest wall. The patient is in a sitting or standing position. The lateral view is taken with the patient supine and the film lateral to the breast; the tube lies perpendicular as close to the chest wall as possible. In both of these views it is essential that the breast be so placed on the film that the nipple is in true profile. This permits evaluation of the degree of retraction of the nipple. Occasionally, in the larger breasts, it is necessary to get an axillary view to include the axillary extension of the breast.

The overall radiographic density of the breast varies greatly, and this is not totally dependent on the size or age of the breast. It is more dependent on the constituent tissues. If the breast contains large amounts of fat which is normally radiolucent, then the overall

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density is greatly diminished, and the fibrous and glandular components, which are denser than fat, stand out clearly.

A tumor within a fatty breast is very clearly outlined. If there is very little fat and a large amount of glandular tissue, as in the virginal breast or in the post-partum breast, then the overall density will be diffusely increased. Tumors, which are generally dense, would, therefore, stand out less well against this background. Slight overexposure of the film is helpful in these cases. Good quality film will portray many of the larger veins. Linear arterial calcification is readily visualized in the older breasts and will outline all of the larger arteries. Fibrous tissue is recognized by its straight, strand-like or web-like nature and is generally directed in a curvilinear fashion from the base of the breast to the subareolar area. The fibrous tissue maintains this normal appearance unless displaced or invaded by tumor. Glandular tissue is recognized radiographically by its cylindrical, elongated, branching densities, starting at the base of the nipple and radiating out to all parts of the breast. These branching radiations are generally undisplaced or uninterrupted except by space-occupying disease or scars from old infection or trauma.

Our main concern in this study is to differentiate benign from malignant disease. If this can be accomplished with reasonable accuracy, then mammography will become a simple and routine procedure of definite value to the surgeon, internist, gynecologist, and also to the general practitioner, who most frequently encounters this problem. Chart No. 1 briefly outlines the basic criteria for the radiological distinction of benign and malignant disease.

Classically, the benign lesion is a round, sharply circumscribed density. There is usually no calcification. If there is fat present in the lesion, such as in a lipoma or a galactocoele, then the lesion will be radiolucent.

The stromal breast markings may be displaced, but they are not invaded. The blood vessels are normal, and there is no localized skin thickening or edema, and there is no nipple retraction. These lesions are also the same size radiographically as they are clinically. This is an important criterion in distinguishing a benign from a malignant lesion, as the malignant process is characteristically smaller by x-ray than by palpation.

Classically, the malignant lesion is irregular in outline, not sharply defined, and occasionally has fine, punctate, sand-like calcifications in it. It is further characterized by having spicules or tentacles that grow out from its main mass, invading the adjacent normal tissue rather than displacing it. Localized skin edema due to blockage of dermal lymphatics and increased venous pattern locally are secondary signs of malignancy and may be evident radiographically when not clinically evident. These are early signs in contrast to nipple retraction, which is a late sign. Also, characteristically, the size of the lesion radiographically is one-half to one-third the size of the lesion clinically.

CHART NO. 1

FINDING	BENIGN	MALIGNANT
1. Skin edema or thickening	None	Positive
2. Nipple retraction	None	Positive
3. Size of lesion by x-ray	Same size as clinical finding.	Smaller than clinical finding.
4. Vascularity	Normal	Increased
5. Calcification	Vascular type only.	Fine grain type calcification.
6. Type of lesion	Smooth, round sharply outlined. Frequently surrounded by radiolucent area. Fatty lesion, such as lipomas or galactoceles are radiolucent.	Irregular, poorly defined, spiculated, lesion, adjacent stromal pattern invading.

Benign tumors of the breast sometimes may be differentiated as to their histological type. For instance, the fibroadenoma is smooth, round, and is usually surrounded by a thin area of radiolucency. The pure adenoma less frequently has this surrounding radiolucency. As stated, galactoceles and lipomas with their high fat content set themselves off as round radiolucencies of varying sizes. Adenosis may be diffuse or localized and is recognized by the enlarged, beaded and branched glandular structure. Scars, either from trauma, surgery, or old infection, show up as collections of fibrous tissue.

The malignant lesions also may be divided as to their histological types. The scirrous carcinoma is ill-defined and shows marked spiculation. The medullary carcinoma is better defined and has only one or two spicules or tails that invade the surrounding tissue. Carcinoma simplex is recognized as disorganized, irregular glandular tissue that lacks the usual branching form. The intraductal carcinoma is an elongated, oval, gland-like structure, poorly localized and poorly demarcated. Punctate or sand-like calcification in the tumor is pathognomonic for this lesion, whether or not an actual tumor mass can be outlined. The fibrosarcoma is well-demarcated, sharp, and does not appear invasive. But it is characterized by extreme density not seen in other lesions, benign or malignant.

Our own experience with mammography has been limited so far to 150 cases. Not all of these have come to biopsy, so that I can only report on the results of the 62 cases that have been biopsied and have pathological reports. These results are summarized in Chart No. 2.

CHART NO. 2

<i>Radiological Diagnosis</i>	<i>Number of Cases</i>
Correct Diagnosis Malignant	19
Correct Diagnosis Benign	33
Total Correct	52 or 83%
False Positive	5
False Negative	6
Total Incorrect	11 or 17%

From this chart, it is evident that the accuracy of radiological prediction of the histology of breast lesions, in our experience, has been 83%.

Upon reviewing our cases, in particular those with false negative or false positive, several factors became apparent. Two of the false negatives and one of the false positives, in retrospect, were errors of interpretation. They occurred during our early experiences with mammography, and were due to lack of experience. Another two of the false negatives, even in retrospect, do not show the lesion and would have to be considered as part of the limitation of the study.

In at least four of those correctly diagnosed as malignant, the clinician did not feel there was any malignant lesion present and, therefore, did not plan to do any biopsy. However, we have not yet picked up a malignant lesion where there was nothing palpable to the clinician.

Along with the increasing accuracy of the x-ray diagnosis in the prediction of the histology of breast lesions, there have developed clear-cut indications for mammography.

Perhaps the most important indication is the patient with multiple lesions, either unilateral or bilateral. Mammography can help decide whether biopsy should be carried out, but more importantly, it can help decide which of the multiple lesions should be biopsied. This additional information is of considerable significance, both to the surgeon and the patient.

With cancer of the breast ranking highest on the list of cancers in the female, it is suggested that mammography might well be considered as a screening procedure in women over forty. This should be given the same importance as the routine, annual chest x-ray or the annual pelvic examination, which now includes a routine Papanicolaou smear for cancer of the cervix. It should be emphasized that, by this method, it is possible to discover malignant breast disease that is not clinically palpable.

As a third indication, mammography is useful prior to biopsy. This at first would appear unnecessary if biopsy is to be carried out anyway. However, if the surgeon has reasonable assurance as to the histological nature of the lesion, it will help him plan both the probable length of the procedure and also the amount of assistance that will be needed. The difference in time between a simple biopsy and a radical breast dissection would be of considerable importance, not only to the surgeon but also the operating supervisor and the anesthesiologist in planning their schedules. It is customary to obtain a chest x-ray before biopsy, and mammography can be done at the same time.

A fourth indication for this type of study is to form a baseline for future studies. At the first examination there may be one or multiple benign lesions, but in the succeeding months a comparative study may be desirable to detect growth of other evidence of malignancy, or to show development of new growths.

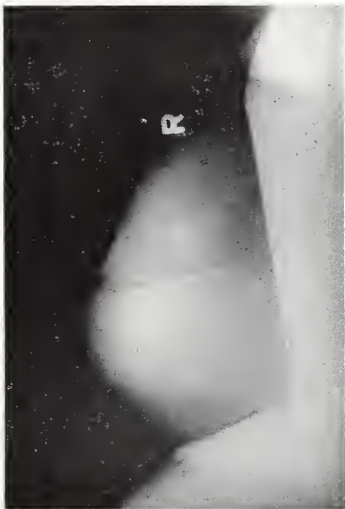


FIG. 1



FIG. 2

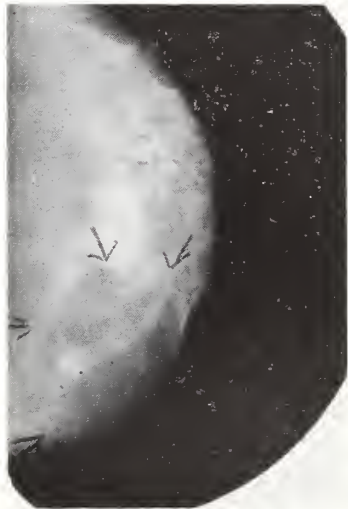


FIG. 3

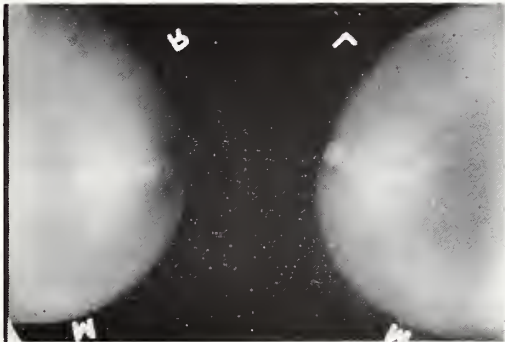


FIG. 4

- FIG. 1. Scirrhous carcinoma upper portion of right breast. Note spiculation, increased vascularity, and nipple retraction.
- FIG. 2. Scirrhous carcinoma in subareolar area. Note spicules, irregular hazy border, and nipple retraction.
- FIG. 3. Three centimeter lipoma lower portion of breast. Note large radiolucent, sharply demarcated area outlined by arrows.
- FIG. 4. Scirrhous carcinoma of right breast in subareolar area. Benign adenomas of left breast in subareolar area.

A lesser indication for mammography lies in the patient who has had unilateral mastectomy for malignant disease. The higher incidence of primary or metastatic disease in the remaining breast has been well documented and its early detection is desirable.

#### SUMMARY

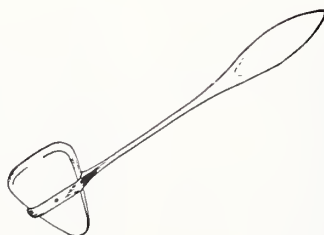
The history, technique, and radiographic findings in mammography have been discussed. The procedure requires no special radiographic equipment and can be satisfactorily performed in any radiological department with a minimum of time and effort. The radiographic examination of the breast has developed into a scientifically reliable, supplementary form of examination with clear-cut indications for its use. It is not intended to replace biopsy.

The author is indebted to Joseph E. Porter, M.D. and Franklin F. Ferguson, M.D. of the Maine Medical Center Pathology Department for their assistance in correlating the x-ray and pathological findings and to Catherine B. O'Connor, R.T. for aid in perfecting the radiographic technique.

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# Infection With *Pasteurella Multocida*

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*Pasteurella Multocida* has been isolated from various human infections. A majority of these have been local infections following the bite of a cat. The carrier rate in the cat, dog, rat and pig is high. Schenk<sup>1</sup> isolated *Pasteurella Multocida* from the nasal passages of 15 of 20 cats, 14 of which were healthy. This organism has also been cultured from the upper respiratory tract of a number of other animals, including a lion<sup>2</sup> which had bitten the hand of a human, causing a severe cellulitis of the forearm. From this wound, *Pasteurella Septica* (*P. Multocida*) was cultured. In most animals, the organism is latent. In humans, however, it may become localized to the soft tissues, or in bone, causing osteomyelitis; or secondly, meningitis; and thirdly, bronchiectasis. According to Swartz and Kunz,<sup>3</sup> only about half of the patients with systemic infections or meningitis present a history of exposure to animals. Most of the infections attributed to an animal have followed a cat bite and about half the humans who developed central nervous system involvement died.

## CASE REPORT

It is felt that the following case should be reported, in view of the potential seriousness of this infection, and that in this case, it was discovered relatively soon. A 7-year-old boy, was suddenly and unexpectedly bitten by a cat. Examination showed three puncture wounds and several scratches on the left upper arm. The puncture wound, which was bleeding moderately, was covered with a dry sterile dressing. That night, he was restless and complained of pain in his entire arm. Seventeen hours later, he was anorexic and lethargic, and complained of generalized muscle pains. Examination of the arm showed moderate edema from shoulder to wrist. Around the wound was a spreading zone of cellulitis about 12 cm. in length. Oral temperature was 101.5°. A thin serosanguinous fluid was exuding from the wound, and this was cultured.

He was started on Declomycin® syrup, 125 mg. t.i.d., and was given 0.5 cc. of tetanus toxoid. On the following day, his temperature was normal and the cellulitis was confined to an area 4 cm. in diameter around the wound. The drainage ceased for two days; then from two of the wounds, drainage was profuse, requiring frequent dressing changes over the course of two more days. The drainage continued for a total of seven days. Declomycin was discontinued after nine days, and healing was complete except for induration deep in the bite wound, which finally became scar tissue after two weeks. The scratches, although severe enough to bleed after the attack, healed in two days without incident.

Cultures were taken from the draining wound on the patient's arm, from the cat ("Herman") who was identified

as the one who had bitten him, and from three other apparently healthy cats in the neighborhood. The original material was planted on blood agar plates and in thioglycollate broth. The colonies on the blood agar grew readily anaerobically at 37° C., and were found to be small Gram-negative bipolar staining rods. Subcultures to endo showed no growth after 24 hours. Fermentation studies made on Cystine trypticase agar, to which sterile carbohydrate solutions were added aseptically, showed acid production, but no gas in dextrose, sucrose and mannitol; lactose and maltose were not fermented. Indol production was positive. Motility tests on Cystine trypticase agar without added carbohydrate were negative. Confirmation of the identity of this organism as *Pasteurella Multocida* was made by Dr. Melvin Gershman, Assistant Professor of Bacteriology, University of Maine, and by Dr. Joseph Schubert of the Communicable Disease Center, Public Health Service, Chamblee, Georgia.

Using the disc method, the organism was found to be sensitive to Chloromycetin®, Declomycin®, Furadantin®, Ganttrisin®, Novobiocin®, Tetracycline®, Erythromycin® and Altabur®, but not to Penicillin, Streptomycin or Bacitracin®. The tests for Penicillin were not repeated.

## DISCUSSION

We believe that the organism isolated in this case was *P. Multocida*. Strains with minimal differences have been isolated from various animals and named according to the animal species from which the strain was obtained. Since the characteristics of most of these strains are so similar, they are now included under the *multocida* species.<sup>4</sup>

This organism was not found to be sensitive to Penicillin. We feel that this observation, whether correct or not, should be mentioned, since previously reported studies<sup>2,5</sup> would indicate that *P. Multocida* is so readily inhibited by Penicillin that sensitivity to this antibiotic might be used as a diagnostic aid in differentiating it from other gram-negative organisms.

This infection in animals can be a most serious problem, particularly in cattle where it may occur in epidemic form, resulting in death from pneumonia and enteritis. Sporadic infection in horses occurs and is known as shipping fever, manifesting itself by an upper respiratory infection and pneumonia.

In man, the three prominent types of infection are: Cellulitis, following the bite of an animal; secondly, meningitis, and according to Meyer<sup>4</sup> this has been most frequently, though not entirely, associated with skull fracture; third, pneumonia, pleurisy and empyema. The most serious of these infections are meningitis and pulmonary infections. Over half of the cases of meningitis have failed to survive, and a large percentage of the organisms have been recovered in cases of respiratory infection who come to autopsy.

Local infections following the bite of an animal are

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one of the most common types. The complications from this, of course, can be serious. It may be followed by septicemia or osteomyelitis. It is therefore important that the cause of the infection be recognized early and prompt therapy instituted. In the case of wounds caused by this organism, surgical drainage should be instituted as well as early antibiotic therapy. When the organism is suspected, penicillin is the treatment of choice, since the organism has been found to be so sensitive to this agent.

It is curious and unexplainable at this time the reason why the organism isolated from the patient described in this case was not sensitive to penicillin in vitro. However, it is presumed that there was probably some error. We were unable to confirm this, since the culture had died out before the error was recognized.

It is also of interest that, in view of the presence of this organism in the oral cavity of all the cats that we cultured, the infection is not more common. This probably is due to two factors: One, there may be an alteration or a loss of virulence of the organism. Secondly, there is probably considerable variation in the susceptibility of humans. The infection is apparently not transmitted from human to human. No adequate explanation has been offered as yet for the pulmonary infection seen in humans.

### CONCLUSION

A case of severe cellulitis in a child due to *Pasteurella Multocida* following a cat bite is described. A similar organism was isolated from the oral cavity of the cat, as well as from several other cats captured in the immediate neighborhood. The infection responded well to Declomycin therapy.

We are indebted to Philip Harvey, M.S. for assistance in the isolation and identification of this organism.

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# Widening Horizons For Medical Radioisotopes

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A time unique in history has come when man possesses nuclear weapons so destructive that he can never use them in anger. He can only demonstrate that he *might* use them by testing them. This very testing provides a means of warfare perhaps not entirely psychological.

While concern about the testing of nuclear weaponry properly increases, it may be helpful for perspective (and sanity) to consider a good which came from the same source and has enjoyed a parallel increase in sophistication and application — the use of artificial radioisotopes in medicine.

A few radioisotopes were in use for research and very limited clinical application in the 1940's. However, it was not until the next decade, and particularly with the Atomic Energy Act of 1954, that this infant medical science had begun to grow out of the universities of its birth into a still increasing number (now many hundreds) of community hospitals across the country. Radioisotope departments have been headed variously by Internists, Radiologists and Pathologists, depending upon individual interests and opportunities in this new field. Actually the ventures have always been interdisciplinary, since a requirement for organization has been a hospital radioisotopes committee. The help and advice of Physicists have been essential. A new breed of trained and responsible technicians has been evolved. Hospital administrations have braced themselves nobly against the problems of financing, space, and housekeeping raised by the sprawling newcomer.

In the past decade a tremendous increase in diagnostic and therapeutic uses of radioisotopes has occurred. This year several hundred thousand people will be tested or treated. Under standards administered by the U.S. Atomic Energy Commission, this will be done with a great margin of safety. Therapy, especially for hyperthyroidism (radioiodine) and polycythemia vera (radiophosphorus), remains on firm ground. Very rapid and exciting advances are taking place in diagnosis where organ scanning and renal function measurement are of particular interest.

Automatic organ scanning systems, which permit quite accurate "pictures" of radioisotope distribution within an organ to be made, are of considerable help in the diagnosis of intracranial, hepatic, splenic and renal lesions as well as (more familiarly) thyroid lesions. A recent symposium in New York which de-

voted two days to scanning techniques attracted five hundred "scanners" from across the nation.

The radioisotope renogram — simultaneous recordings of the rates at which the kidneys handle radioisotope-labeled materials — promises much in the differential diagnosis of renal disease, particularly when a significant unilateral vascular lesion is present.

These tests are being added to an armamentarium which has for some time permitted such valuable studies as those of thyroid function, blood volume (total and red cell mass), vitamin B<sub>12</sub> absorption (Shilling test) and red blood cell survival. The list is not complete by any means.

In our own State, progress has been gratifying. Attempts to blend Yankee conservatism with a "nor yet the last to cast the old aside" attitude have perhaps been justifiable in these developmental years. Eight years ago in a small basement room in a Portland hospital the first diagnostic dose of radioiodine (I<sup>131</sup>) was given. An earlier article reviewed the technique.<sup>1</sup> Nearly two thousand doses later we consider this technique, somewhat modified, reliable when its limitations are recognized. Seven years ago the first therapeutic dose of I<sup>131</sup> was given, the first of nearly three hundred.<sup>2</sup> We continue to regard this as the treatment of choice in the patient over 45 with diffuse thyroid hyperplasia. Other diagnostic and therapeutic measures have been made available as their value has been established.

Meanwhile excellent radioisotope laboratories have been developed to provide needed services in all of the larger hospitals in the State. We have enjoyed a cordial liaison through the years. It seems certain that growth is inevitable for all of us.

I<sup>131</sup>, a gamma and beta-emitting radioisotope of approximately eight days' half-life, is produced for mankind in two major ways: (1) in uncontrolled amounts of unpredictable destiny in a nuclear explosion and (2) in controlled amounts of predictable destiny from a peaceful nuclear reactor.

Our hope and prayer is that if and when you get your dose, it will be from (2) rather than from (1).

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# Some Observations On Medical Education In Maine

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Changes must be made for medical education in Maine. New ideas and new concepts must be tried in house staff training, as well as educational programs for physicians in practice. The Maine Medical Center is attempting to devise a new approach to this problem. Ideally, a medical school would act as a fulcrum for a new educational project. Any proposal should therefore be made with the consideration of a medical school for Maine as the frame of reference.

The influence of a medical school is important to physicians in the area in which it is located. With its associated hospitals, a school offers both specialized and comprehensive medical service to patients. Through academic tradition and support to research, a school maintains highly specialized equipment and trained personnel, who respond as a team operating the highly complex equipment required of modern medicine. The combination of the medical school and its associated hospitals influences the day to day patient care in a still broader sense by providing education, training and research opportunities for physicians in practice. It maintains quality graduate training programs, and either maintains or stimulates continuing medical education for physicians. Even in areas where good medical care is known to exist, a medical school further promotes improved patient care.

Maine has not had a medical school since Bowdoin Medical College closed its doors in 1922. Measured either by time or distance, the closest four-year medical schools to Maine are located in Boston. Their influence at least partially stimulates phases of patient care and professional growth in the southern part of the State, but it may be asked "Is this influence sufficient? Is it sufficient for southern Maine as well as the entire State?" These questions are difficult to answer categorically. While it is possible that the influence is adequate for some parts of southern Maine, it is almost certain that it is not adequate for the central and northern portions of the State. In these areas the distance is too great.

Do the physicians and the people of Maine want a medical school? From the information one gathers through personal conversations, there is no doubt that those who have considered the proposal carefully say "yes." The question then becomes: Can Maine afford a medical school? This is more difficult to answer. The

report of the President's Commission on the Health Needs of the Nation, offered through the Surgeon General's Consultant Group in Medical Education — "Physicians for a Growing America" — states that the approximate cost of a two-year medical school is fifteen million dollars and for a four-year school fifty-million dollars. This is a lot of money for a new institution, and for many states, especially states like Maine, where economy does not provide sufficient revenue to pay for these projects, even when financing is done by issuing bonds. It is rumored that the need for physicians and medical schools is so great that the Federal Government may soon assist in the development of these highly expensive institutions, but even with to-day's thinking, and including anticipated Federal support, these goals may be achieved slowly in Maine as elsewhere.

If these goals cannot be achieved within the next year or two, when can they be achieved? It takes about five years to complete a medical school from the planning stage to an operating institution. Depending on whether it is a two or four-year school, it takes that much longer to turn out students. As of today, Maine has not formally decided whether it will have a medical school. It has not even decided whether it would be a two-year or four-year school, where it should be located, and what clinical facilities it will need. These and many other questions have been discussed but remain unanswered.

Can steps be taken to provide, at least in some measure, the beneficial influence of a medical school, at least until one is established? If steps can be taken, what are they? In Maine, and similar areas, a new kind of medical institution needs to be created. The community hospitals have long been considered by some as a place to train interns and residents, but they have never primarily been considered schools in the strict sense of the word. Since Maine does not have a medical school and accepting the fact that the influence of education and training is beneficial to the patient and the public alike, the three community hospitals approved for house staff training should be developed as training centers, using intern and resident training programs as their first goal, and continuing education for practicing physicians as their second goal. The foundation of these new teaching facilities could be an experiment in medical education providing education for medical students, interns and residents, as well as physicians in practice. From the start these hospitals

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should have a medical school affiliation. If subsequently a medical school is developed in Maine, one or more of these institutions may choose to change their affiliation. In the meantime they can provide better care for patients, a new form of education for physicians and replenish the depleted supply of physicians in the State.

If these "teaching centers" are to be successful, a lot of work and some sacrifice must be made. The co-operative efforts of organized medicine within the State are needed. Plans must be formulated in each of the three hospitals with approved training programs, and

these plans must be extended into the County and State Societies. The Council on Medical Education and Hospitals of the American Medical Association is interested in furthering this type of physician education. There is urgency for co-operation for both medical education and medical practice. As physicians we must work together on projects which make the best of the existing opportunities if we are to cherish the practice of medicine in a free society. Here is offered one of the many challenges facing medicine today.

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## Changing Concepts Of Malpractice Laws\*

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Having been identified with the Medical Advisory Committee in the State of Maine for more than 20 years, I have been extremely interested in the problem on the state level of the changing concepts of Malpractice Laws. I am pleased to have this opportunity to discuss some of these changes with you.

If I may become sentimental for a moment, I wish to state here, categorically, that ours is an old and honored profession. A profession to which each and every individual is a dedicated person, otherwise he would not be practicing the healing arts. A profession which knows no forty hour week, no fringe benefits, no time and a half or double time; a profession which over the years, has been dedicated to one goal and one goal *only*, service to its' fellow man. I would like to state here that I know of no profession or trade or human endeavor which over the years has worked so diligently and so industriously to put itself out of business as the Medical Profession. This is a record of which we all can be justly proud. No other profession, and I say this unequivocally, can pretend to approximate this record. Therefore, to me it seems rather paradoxical that with a profession which has worked so hard to increase its standards, to eliminate the diploma mills, to provide the best medical care in the world, that we have become the whipping boy of the "do-gooders," the "social planners" and for the past few years, fair prey for the individual and his attorney who is after the "*fast buck*." I want to impress upon you, that Malpractice Laws are rapidly changing and, unfortunately, mostly in the

favor of the plaintiff and his attorney. As you all know, our code of law in this country is derived from English Common Law. The two types of law with which the average person is familiar are statutory law which is made by Legislatures and Assemblies, and common law. A statutory law remains in effect until a new law is passed which abolishes it or takes its place. The second type is composed of large groups of rules developed by the early courts and are not contained in Legislation. This body of rules is known as the Common Law and is interpreted by the courts. In English speaking countries court decisions commonly rely heavily upon previous court customs or precedent. Therefore, malpractice actions are governed by the second type which is known as Common Law. Thus the evolution in practice of these new theories of law in regard to negligence cases against the physician.

We might ask ourselves here, "Why are malpractice cases becoming more prevalent?" I suppose there are many answers. First of all, during the past thirty years we have been drifting down the road of the Welfare State and are fast becoming a nation of materialists. Frequently it may be on the basis of an argument with the doctor about fees, a common factor; or the patient may be dissatisfied because the surgeon failed to bring about a miracle and often suits are filed hoping to scare the doctor into cancelling his bill for professional services. In other cases the suit prone patient is well educated in the matter of a "fast buck" and he doesn't need much in the way of provocation to start a legal action. Frequently, action may be started by an unsolicited comment from one physician relative to the treatment or result of another physician. Be that as it may, the general public is becoming more suit conscious than ever before.

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At this point I believe it is important that we define "Malpractice." Malpractice might be defined as "improper professional action or conduct as for reprehensible ignorance." I am sure the legal definition, however, would be of much more length in order to cover errors of omission and commission. Be that as it may, there is not a physician in Maine who can say, "It can't happen to me." One of every four members of the A.M.A. in New York and California and one in every seven in the United States has been sued for malpractice. From these statistics it would appear that every time a physician treats a patient he must keep one eye on the patient and the other on the court house down the street. That being true, let us for the moment consider some of the ingredients necessary for malpractice action. In my opinion, I believe there are four important ones to be considered; a) breach of contract; b) breach of warranty; c) liability without fault; and d) informed consent. For instance, if you tell an anxious patient that your treatment will get him well and it doesn't, you are liable for a "breach of contract" suit. If you attempt to repair a hernia and tell the patient you can fix it and it recurs, you may be facing a "breach of warranty" action. If it so happens that the results of any given case are bad even though the case has been handled skillfully it becomes "liability without fault," which sounds rather paradoxical. I fail to see how one can be *liable* if there is no *fault*. In other words, "you gave the proper treatment, doctor, but you got a bad result therefore you must pay damages." This is an interesting concept of law and justice. When one considers this "liability without fault" as a concept of law, I can well understand why the Goddess of Justice has a blindfold. The fourth ingredient or item, "informed consent," is a rather recent concept which is gradually spreading throughout the country and with which you are going to come in contact. During 1960 the Supreme Courts in Kansas and Missouri created a new law in which they held that it is for the jury to determine whether the administration of treatment was given with the "informed consent" of the patient; if it was not, then the physician is guilty of malpractice. In normal circumstances the adult patient must be the final arbiter as to whether or not to undergo treatment or surgery even where his life is imperiled without it. Exceptions are emergencies where it is impossible or impractical to obtain the patient's consent or the consent of someone authorized to assume such responsibility. The general rule prohibiting the performance of an operation without the consent of a patient extends to operations different in nature to that for which the consent was given and to operations involving risks or results not contemplated.

Now, we might ask ourselves, "What is informed consent?" The consent given by the patient or someone authorized to act in his behalf must be an "informed consent" with an understanding of what is to be done and the risks involved. The consent may be invalid if

a) the act consented to is unlawful; b) the consent was given by one who has no legal right to do so; and c) if it was obtained by misrepresentation or fraud. In the absence of clearly specified prohibitions on the part of the patient, the physician should be privileged to perform such surgery within the operative field as is justified by the prevailing medical opinion. The patient in a teaching hospital and his attending physician are in a medico-legal situation which differs in some respects from that of a patient being treated in a non-teaching hospital. A teaching hospital includes not only those institutions which are operating in connection with a medical school but also the unaffiliated hospital that maintain an American Medical Association approved teaching program for interns and residents. Teaching hospitals are obligated, in order to maintain an A.M.A. approved program, to provide sufficient clinical material necessary for the adequate instruction of interns and residents. Therefore, when a patient enters a teaching hospital, and knowing its nature and purpose, there is an implied understanding on his part that he thereby consents to participate in the teaching program. It is desirable that the physician explain to the patient that the teaching function of the institution contributes to the high standard of medical care. From the purely legal standpoint the attending physician's consent is not required to permit examination of the patient by the Director of Medical Education in a teaching hospital. The patient's consent is all that is necessary. Furthermore, if the patient consents to an examination by the Director of Medical Education this constitutes consent for legal purposes. A private patient in a teaching hospital should not be subjected to unnecessary tests which would cause him either discomfort or added expense merely to satisfy the needs of the teaching program. Although the law is not precisely defined the holdings of recent cases involving lack of consent may make the physician a frequent target for malpractice claims whenever a bad result occurs. Since the gist of the action does not involve negligent treatment but negligence in failing to explain the hazards to the patient, the claim of alleged lack of "informed consent" may become attractive to those attorneys who seek new "theories" of liability against the physician. Under the circumstances the physician must be prepared to prove in court that he explained the risks involved to the patient whenever surgical, therapeutic or diagnostic procedures involve more than the hazards which the patient might normally expect. The physician's best protection is to inform the patient fully regarding any unusual risks that may be involved and to insist upon a consent in writing in which the patient acknowledges this explanation. Because of the importance of this "informed consent" the A.M.A. Law Department provides a general form of consent covering a number of procedures. If this form is used the physician should cross out any sections that do not apply. The consent form should also include the patient's acknowledgement

of the explanation by signing the consent form with the usual witnessed signatures and with the date and place. Documents should be prepared in three copies, one for the patient, one for the hospital record and one for the doctor's office file. In this way everyone is thoroughly informed and protected. Many of us in the past, and I suppose many of us still are, accepting the patient's oral consent or rely on the operative permit which the patient signs upon admission to the hospital. I wish to stress here for your own protection that the oral consent or the signed hospital form is not *sufficient protection* in case of a *malpractice action*. Informed consents should be signed in your office in the manner stated above. This informed consent which necessitates discussion of the element of risk or risks will require a certain amount of discretion and tact consistent with the full disclosure of facts necessary for an "informed consent." As we all fully appreciate many patients contemplating a surgical procedure are frequently emotionally disturbed and may be tremendously traumatized psychologically by the explanation of the risks involved necessitated by this doctrine of "informed consent."

This reminds me of a cartoon which I saw recently; the doctor explaining "informed consent" to a patient, telling her that if she took the pills she might have a reaction, might break out into a rash, possibly experience some G. I. bleeding and after going into the lurid details saying, "Now, damn you, do you want to take these two Aspirin?"

Before closing this discussion, I would be quite remiss if I did not mention the doctrine of "Res Ipsa Loquitor." This doctrine of Res Ipsa as applied to medical malpractice has raised its voice in court again after a deceptive silence. Originated in England in 1863 the doctrine was first applied to a medical negligence case in Alabama in 1923. It had an alarming resurgence in California several years ago but was not generally accepted in the courts of other states. Now, in a recent malpractice case before the New York County Supreme Court "Res Ipsa" appears to be making inroads on the East Coast. The basic theory of our law is that *he* who *accuses* must *prove*. In medical malpractice cases this means prove by expert medical testimony to the jury, which alone is empowered to determine all disputed questions of fact. It can have no opinion on complicated medical matters unless furnished that opinion by doctors themselves testifying in the case. Nothing is more *fundamental* to law than that a jury must not be permitted to *speculate*. Under our law it is just as pernicious to submit a case to a jury and permit the jury to speculate with the rights of citizens when no question for the jury is involved as to deny a citizen his trial by jury when he has a right. There long has been an exception to the rule of "he who accuses must prove" inherent in the rationale of the rule itself, namely, when the act complained of by the patient is so simple as to be within the lay knowledge of the average citizen juror. For example, a) hot water bottle burns; b) foreign

bodies left in tissues etc. In such cases the jury can form an intelligent opinion without the benefit of expert medical testimony. The *second* exception to the *fundamental* rule that he who *accuses* must *prove* which in recent years has been more and more applied to malpractice cases is known by the Latin phrase, "Res Ipsa Loquitor."

Originally the offspring of a casual comment of Baron Pollack during legal argument the phrase translated means "the thing speaks for itself." Around this phrase there has evolved a legal doctrine that has been severely critized; "*It adds nothing to the law, has no meaning which is not more clearly expressed in English and brings confusion to legal discussions. It does not represent a doctrine, is not a legal maxim and is not a rule.*" It is simply this, negligence may be proved by circumstantial evidence. One type of circumstantial evidence to which the courts have given the name Res Ipsa Loquitor arises when a) the accident does not ordinarily occur in the absence of someone's negligence; b) it is caused by instrumentality within the exclusive control of the defendant and c) the possibility of contributing conduct which would make the plaintiff or patient responsible is eliminated.

Res Ipsa principles have been applied to cases of falling objects, explosions, railroad derailment, etc. The extension of the doctrine would seem to make Res Ipsa Loquitor merely a "rule of sympathy" rather than a "rule of law." What is alarming is a recent tendency in some courts to extend this perversion of Res Ipsa Loquitor in medical malpractice cases. Any physician who fails to cure or obtains an untoward result with no evidence of negligence faces the prospect of running the gauntlet of jury speculation. Therefore, under these circumstances the doctrine of "Res Ipsa Loquitor" is nothing more than "liability without fault." I would like to quote an excellent opinion on the applicability of Res Ipsa Loquitor doctrine in malpractice cases, written by William Howard Taft, then a Federal Circuit Judge and later President of the United States; "The physician is not a warrantor of cures. If the maxim Res Ipsa Loquitor were applicable and the failure to cure were held to be evidence, however slight, of negligence on the part of the physician or surgeon causing the bad result, few would be courageous enough to practice the healing art for they would have to assume financial liability for nearly all the ill's flesh is heir to."

It is easy to accuse, it is difficult to defend. The *safeguard* of the *law*, that *he* who *accuses* must *prove* and in malpractice cases must prove actual negligence by expert testimony is sound and should be preserved. To do otherwise is to force the medical profession into a role of insurers, a burden which it cannot and will not bear without the resultant loss of useful techniques and retardation of medical science. We have reached the point where we ask ourselves, "What can we do about all this?" I would like to suggest several positive courses we may pursue: 1) *First* on the list I would

place *Medical Records* — both office and hospital with especial emphasis on adequate Progress notes. Your records are invaluable in contesting a malpractice action. 2) Next I would add the "Informed Consent" portion of the record especially in *surgical* cases and *others* which involve the *newer therapeutic procedures*. 3) More frequent consultations, especially when a case isn't doing well or when the patient or his family appear dissatisfied. 4) In traumatic cases order adequate x-rays despite the expense. 5) Increase your malpractice coverage to at least \$100,000 — \$300,000. 6) When threatened with suit, immediately notify your Insurance Carrier and your legal counsel. 7) Attempt to educate the patient and public reincreased x-ray, laboratory and other charges due to increase of malpractice cases. 8) If you are advised by your Legal Counsel and

your Committee to go to court to defend an action, please do not be afraid to do so in spite of so called adverse newspaper publicity. Personally I would much prefer to take my chances with a "jury of my peers" rather than become the victim of legalized extortion and settle behind closed doors. At least, in the State of Maine we are fortunate that the Contingency Fee which many states permit is *unethical*. Canon 13 of the Professional Ethics of the Maine State Bar Association expressly disapproves the Contingent Fee. Our colleagues in New York State are not so fortunate and the amount of the contingency fee is 50% of the award. To put it bluntly, in New York State, the plaintiff and his attorney are in business together.

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## Report On Research At The Maine Medical Center: Annual Meeting, 1961

HAROLD L. OSHER, M.D.

When I was told that a report on research was to be given at this meeting, it struck me as significant that in a community hospital where a few years ago, there was little or no research activity and no research committee, a report from that committee should be one of a select few presented at this annual meeting. I was able to think of several possible reasons for this. One is that doctors seem to be fascinated by two things; the new (new diagnostic methods, new drugs, etc.), and the odd or esoteric or rare; research at the Maine Medical Center is relatively new, and at least in the minds of some, is odd and esoteric and rare. Then I was cheered by the thought that at last it was recognized that research is a good thing and important enough for the staff to know more about; my cheer was somewhat dampened by the nagging suspicion that the motivating thought might have been that there is skullduggery afoot and it's high time for an accounting!

But whatever the reason for this report, it might start by attempting to answer the questions which are on your many minds. These are: Why on earth is the Maine Medical Center getting involved in research? Isn't this a community hospital whose primary function is the care of the sick? It is perhaps understandable for us to depart from this primary function to the extent of providing postgraduate training for house officers and even practicing physicians (if this does indeed constitute a departure), but isn't research the province of

the university hospital or medical school or Rockefeller Institute or the National Institutes of Health? Now these are all good questions and I think I can best answer them by quoting from the 1959 annual report of the Research Committee, which some of you may have overlooked.

The modern hospital must not only provide patient care of high quality, but must also serve the community as a center of medical teaching and research. This fact is well known by hospital administrators and trustees, and is becoming so by a public increasingly informed and concerned about health matters. As stated in the American Foundation's midcentury survey of medical research, "modern hospitals are medical teaching centers, good or bad, whether they wish it or not."<sup>1</sup>

Dr. Randall G. Sprague of the Mayo Clinic, commenting that to oppose research today would be like opposing virtue, has given several cogent reasons why "research is an essential ingredient in institutions which aspire to the best in care of patients and in teaching,"<sup>2</sup> They are:

1. Research training equips the young physician to gather, integrate and analyze data in the solution of medical problems, making him a better practitioner if not a leader in medical science.
2. The prestige of the hospital is closely related to that of its staff and of the physicians it trains, as measured by their contributions to medical pro-

gress as well as their proficiency in the care of patients. (It might be added that the enhanced prestige acquired in this way undoubtedly facilitates the obtaining of desirable interns and residents).

3. Patient care is improved through application of increased up-to-date knowledge, achieved through scientific study of disease and critical evaluation of available diagnostic and therapeutic methods.
4. Hospitals have a *moral obligation* to contribute to medical knowledge through research and study.

I might add a fifth reason which applies particularly to this hospital; and that is, that certain patient care facilities would not be available to us if it were not for the funds and personnel provided by research programs; the artificial kidney is a good example of this.

Now that we know why we're doing it, what are we doing? Because of time limitations I can only tell you briefly about the various projects going on, and I apologize for those that I may omit. I refer you to this year's annual report of the Research Committee for further details. Most of you have heard of the so-called "Nelson Project"; Clifford Nelson Ph.D. has for years been carrying out studies designed to characterize and quantify the heart vector or the electrical forces of the heart; this work is highly regarded by those able to appreciate it, and the results have been published in some of the better journals and presented at national and international meetings. The work has now reached the point where it may soon have practical application in clinical vectorcardiography, and some of the technics and instruments developed by Dr. Nelson can be used for clinical investigation. I dwell on this because some of us like to see a practical application for the result of even basic research, and also because of the prestige and recognition this work has brought to our hospital.

Dr. Jerome Tichy is studying the sodium and potassium binding capacity of basic amino acids and myocardial protein. Again, this is basic research with potential practical significance.

In the pathology department Dr. Joseph E. Porter has developed technics for studying human chromosomes. In the G.I. research laboratory Dr. Irving J. Poliner is evaluating anticholinergic drugs and investigating radioactive iodine secretion by the stomach. In the heart station Drs. Manu Chatterjee, William Austin and Peter W. Rand are studying metabolic alterations in hypothermia and total body perfusion in connection with open heart surgery, which is in itself actually a clinical research and development program; the same group is also studying hemodynamics in hypertension and shock, indicator dilution methods and instantaneous multi-lead electrocardiograms; Dr. Stanley G. Dienst is investigating organic acids in uremia; Dr. Austin will shortly initiate studies of radioactive renograms; Drs. Harold L. Osher and Niles L. Perkins, Jr., are investigating heart

sounds and murmurs by means of a miniature microphone inserted into the heart on the end of a catheter; and Dr. Osher is evaluating the effect of Triparanol or MER-29® on anticoagulant therapy. In other areas, Dr. Stanley E. Herrick, Jr., is evaluating the treatment of metastatic breast cancer and Dr. John R. Lincoln has completed studies of antiemetic drugs in surgical patients.

I think the fruitfulness of these and other projects is illustrated by the programs of the forthcoming regional meetings of the American College of Physicians and American College of Chest Physicians, which list reports on the above projects of Drs. Austin, Rand, Chatterjee, Osher and Perkins, as well as papers by Dr. John F. Gibbons on cineradiographic studies of esophageal lesions, Dr. Emerson H. Drake on surgery of cardiospasm, Dr. Gisela K. Davidson on tuberculosis and lung cancer and Dr. Clement A. Hiebert on a new and original method for correction of esophageal reflux.

As you can see, we have a growing research program, and it is gratifying to see it acquire a broader base as more services and departments of the hospital are taking part. Along with this expansion, you can imagine that problems have arisen; these have been largely in the areas of administrative policy, space and finance. To deal with administrative and policy matters, a Joint Research Committee has been formed consisting of three trustees, the director and assistant director of the hospital, and two members of the staff, the president, Dr. Maltby, and the chairman of the staff Research Committee. The space problem has been a difficult one, and has been helped considerably by the action of the Joint Research Committee in making available the old house officers quarters for research purposes. Efficient utilization of this area now awaits the funds for necessary renovation and equipment; we hope these will soon be forthcoming. This brings us to the third problem, finance, which has also been and will undoubtedly continue to be a difficult one. Support of all research to date has come from sources outside the hospital; The National Heart Institute, the Maine Heart Association, the Pfeiffer Foundation, and private gifts; each has included an allowance for hospital overhead. The Joint Research Committee has felt to date that patient care income should not be used to support research, and that research should not increase the cost of patient care. We are hopeful that some day an endowment fund for support of research will lessen the economic squeeze and uncertainty of dependence on year-to-year grants. It goes without saying that the future of research here depends largely on the medical staff; if the research program is to grow and succeed, it is the staff who will initiate it, carry it on, and support it or find support for it. We are fortunate in having an enlightened and progressive administration and board of trustees who have demonstrated their willingness to help us all they can.

Finally, I would like to tell you about some recent activities and decisions of your representatives on the

Research Committee, as noted in our interim report to the executive committee. Because of the increasing volume and scope of research activity, the committee is now holding regular monthly meetings: it is taking a more active role in the stimulation and encouragement of research as well as in its supervision and control; it plans to make regular reports to the staff and executive committee in order to keep you informed and to avoid misconceptions; along these lines, the Scientific Committee has accepted the suggestion of the Research Committee to invite research workers to present their findings periodically at the scientific sessions of the monthly staff meetings.

Now this has been a rather sketchy account of a large subject, but it's all that time permits. As indicated earlier, further reports will be forthcoming.

#### REFERENCES

1. The American Foundation: Medical Research: A Mid-century Survey. Vol. 1, American Medical Research in Principle and Practice; Vol. 2, Unsolved Clinical Problems in Biological Perspective, Boston, 1955, Little, Brown & Company, Vol. 1, p. 560.
2. Sprague, Randall G.: The Essentiality of Clinical Research: Some Comments on its Role in Institutions which Practice and Teach Medicine, J. Lab. and Clin. Med. 49: 2-6, 1957.

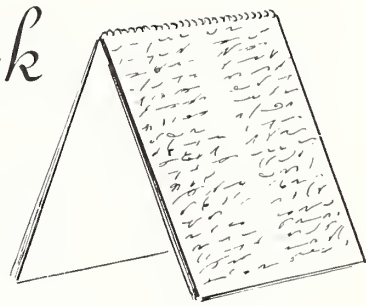
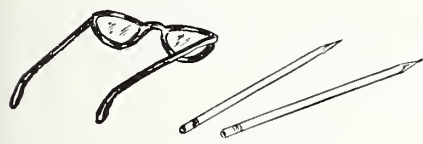
131 Chadwick Street, Portland, Maine

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### The Cold Bureaucratic Hand And Mind

The (Kefauver) Committee spent long and dreary hours bedeviling and belaboring the manufacturers for the way in which they spread the word concerning their new prescriptions. Admittedly, it's an expensive process. It has to be. They must tell the physician, individually, for he alone will do the prescribing. The United States medical profession is not a mass audience. It is 150,000 practitioners, each one dealing with individual cases of life and death. The doctor is told personally about new drugs, not by a salesman, but by a skilled and trained specialist. A new antibiotic, after all, is not a new flavor chewing gum. The physician must know such facts as dosage form, composition, indications, contraindications, and side effects, if any. The system not only introduces new drugs in the market place. It also happens to serve and protect the public interest — by which I mean the individual patient. But in any case, drug promotion is an industry matter far better handled by private industry, than by the stultified, cold bureaucratic hand and mind. — Senator Roman L. Hruska to annual meeting of the Drug Trading Company, Ltd., Toronto, Canada, May 2, 1962.

# From the Secretary's Notebook



## 109th Annual Session of the Maine Medical Association House of Delegates

Forty-seven members of the House of Delegates were present at this 109th annual session at which many timely and interesting subjects were discussed. The first meeting, which was called to order by Ralph C. Stuart, M.D., President-elect, on Sunday, June 17, 1962 at 10:15 a.m., adjourned at 1:00 p.m. The afternoon session convened at 3:30 p.m. and adjourned at 6:00 p.m.

### Election of Speaker and Vice-Speaker

First on the order of business was the election of a Speaker and Vice-Speaker as approved at the Interim Session of the House of Delegates in April.

Linus J. Stitham, M.D. of Dover-Foxcroft, was elected Speaker of the House of Delegates and Robinson L. Bidwell, M.D. of Portland, elected Vice-Speaker.

Following considerable discussion and a roll call vote, it was voted that the Speaker and Vice-Speaker assume office at the conclusion of the annual meeting at which they are elected to serve for one year.

It was further voted that for this session only, Dr. Stitham assume his duties in the capacity of Speaker of this House of Delegates. (Dr. Stitham then assumed the chair, as Speaker of the House of Delegates, for this session.)

### Reference Committees

Three Reference Committees, consisting of the following members, were appointed by the President-elect, Dr. Stuart.

Reference Committee No. 1 — Robinson L. Bidwell, M.D., Portland, Chairman; Carl E. Richards, M.D., Sanford; and Waldo A. Clapp, M.D., Lewiston.

Reference Committee, No. 2 — Albert P. Royal, Jr., M.D., Rumford, Chairman; John D. Denison, M.D., Gardiner; and George E. Sullivan, M.D., Fairfield.

Reference Committee, No. 3 — Arthur N. Lieberman, M.D., Bangor, Chairman; John W. McAllister, M.D., Lubec; and Elizabeth E. Williamson, M.D., Blue Hill.

### The Budget

A motion by Charles R. Glassmire, M.D. of Portland, that the Budget for fiscal year 1963, as drawn up by the Budget Committee (Doctors Thomas A. Martin, Charles W. Eastman, and John F. Dougherty) and presented at the Interim Meeting of the House of Delegates, be adopted was duly seconded and carried unanimously.

Estimated income from January 1, 1963 to December 31, 1963 from State Dues, Journal advertising, Subscriptions, Exhibit Space Rentals, and miscellaneous is \$69,420.58.

Approved expenditures total \$74,340.00 as itemized below:

Association	
Office	
Salaries:	
Executive Director	\$11,000.00
Secretary-Treasurer	3,500.00
Stenographers	6,240.00
Travel — Exec. Dir. & Sec. Treas.	1,200.00
Supplies, tel., rent, payroll taxes	5,500.00
Equipment	500.00
General:	
President's Expenses	1,000.00
Annual Session & Int. Meet. House of Delegates	5,000.00
Committees:	
Medical Advisory (Legal Counsel)	1,000.00
Legislative Counsel	1,500.00
Standing & Special	1,500.00
Delegates:	
American Medical Ass'n.	1,200.00
N. England & New Brunswick	400.00
New England Council Dues	150.00
Fall Clinical Session	500.00
Annual Roster	300.00
Woman's Auxiliary	400.00
Journal:	
Printing & Plates	20,000.00
Travel	250.00
Office:	
Salaries:	
Editor	2,500.00
Secretary-Treasurer	3,000.00
Stenographer	3,400.00
Supplies, postage, rent, payroll taxes	1,800.00
Insurance	100.00
Retirement Fund	2,400.00
Totals:	\$74,340.00

### Annual Reports

Reports of Standing and Special Committees, which had been submitted prior to the meeting, were included with the  
*Continued on Page 205*

## Report Of Delegate To A.M.A. House Of Delegates Annual Meeting, Chicago, Illinois, June 24-26, 1962

I arrived in Chicago on the evening of June 23, and from 7:00 A.M. on Sunday, June 24 until I left for the airport at 11:30 A.M. on Thursday, June 28, my time was taken up completely in attending official meetings relative to the House of Delegates. I also served on a Reference Committee for Amendments to the Constitution and By-laws.

On Tuesday evening I represented Dr. Ralph C. Stuart at a reception for state presidents. This was an innovation held in connection with the inauguration of the A.M.A. president. It was a very impressive affair and it was with regret, I am sure, that Dr. Stuart was unable to attend.

Usually the president-elect of the A.M.A. is a physician who has been connected with the A.M.A. as a delegate, has served on committees for several years and has been a trustee, but this year a younger physician, who is relatively new to the A.M.A. was chosen president-elect. Dr. Edward R. Annis of Miami, Florida, chairman of the A.M.A. National Speakers Bureau and well known spokesman in the campaign against the King-Anderson Bill, was chosen president-elect. Dr. Annis will become president at the June, 1963, annual meeting in Atlantic City, succeeding Dr. George M. Fister of Ogden, Utah who assumed office at the Tuesday night inaugural ceremony.

Final registration figures at the meeting reached a total of 42,643, including 14,092 physicians.

### HEALTH CARE FOR THE AGED

The House received 17 resolutions expressing full support of the Kerr-Mills program and firm opposition to the King-Anderson type of legislation.

Dr. Fister in his inaugural address said, "We will not compromise with those who regard medical care problems as playthings in the game of politics — gimmicks to attract the votes of the gullible."

### MEDICAL DISCIPLINE

To implement one of the major recommendations made by the Medical Disciplinary Committee at the June, 1961 meeting in New York, the House approved a change in the by-laws under which a proposed Section 1 B of Chapter IV is quoted:

"In addition to such disciplinary action as may be taken under the constitution and by-laws of the component society and constituent association to which the member belongs, or when a state medical association to which a member belongs requests the A.M.A. to take disciplinary action, or when at the request of the A.M.A. the state association to which the member belongs consents to disciplinary proceedings by A.M.A., the Judicial Council, after due notice and hearing, may censure him, or may suspend or expel any member of the A.M.A. from A.M.A. membership only for an infraction of the Constitution or these By-laws or for a violation of the Principles of Medical Ethics."

### A.M.A. BOARD OF TRUSTEES

The House approved a report of the Ad Hoc Committee on the Board of Trustees which recommended that the size of the Board be increased from 11 members to 15 members. This will be accomplished by adding three elected members and by including the immediate past president of the Association for a one year term. The House also accepted a committee recommendation that set the term of office for elected Board members at three years and limited the number of terms to three, for a maximum total of nine years service. To implement the House action, the Council on Constitution and

By-laws submitted changes in the Constitution and By-laws for consideration at the 1962 Clinical Meeting.

### AMERICAN BOARD OF ABDOMINAL SURGERY

A study report from the Council on Medical Education and Hospitals, recommending that recognition should not be granted to the American Board of Abdominal Surgery as a specialty board, was approved by the House. In accepting the Council report, the House also declared its disapproval in principle of establishing specialties which are based largely or wholly on or arbitrarily defined anatomical region of the body.

The study, which was carried out under instructions from the House of Delegates at the 1961 Clinical Meeting, concluded that the present contribution of the American Board of Abdominal Surgery to the Advancement of surgery and the betterment of public health is inadequate in many important respects. It also concluded that the American Board of Abdominal Surgery does not offer significant potential for the advancement of surgery and the betterment of public health.

### AMERICAN COLLEGE OF SURGEONS

In considering a Board report and four resolutions involving surgical assistants and relations between the A.M.A. and the American College of Surgeons, the House declared that the adoption and interpretation of the Principles of Medical Ethics is the prerogative and duty of the American Medical Association. It also restated the Association's June 1961 policy statement in the following manner:

- "1. Each member of the A.M.A. is expected to observe the Principles of Medical Ethics in every aspect of his professional practice.
- "2. Each doctor engaged in the care of the patient is entitled to compensation commensurate with the value of the services he has personally rendered.
- "3. No doctor should bill or be paid for a service which he does not perform; mere referral does not constitute a professional service for which a professional charge should be made or for which a fee may be ethically paid or received.
- "4. When services are rendered by more than one physician, each physician should submit his own bill to the patient and be compensated separately whenever possible.
- "5. It is ethically permissible in certain circumstances, however, for a surgeon to engage other physicians to assist him in the performance of a surgical procedure and to pay a reasonable amount for such assistance. This principle applies whether or not an assisting physician is the referring doctor."

### MISCELLANEOUS ACTIONS

The House also disapproved a suggestion that the Council on Medical Education and Hospitals be replaced by two separate councils on undergraduate and graduate medical education.

It referred to the Board of Trustees a proposal that at least six members of the Council on Medical Education and Hospitals shall be engaged primarily in the private practice of medicine in hospitals without a medical school affiliation and that no more than four members may be salaried personnel of a medical school or university.

It approved a recommendation that A.M.A. meetings be scheduled as follows: Annual Meetings — 1966, Chicago; 1967, Atlantic City and 1968, San Francisco, and Clinical Meetings — 1965, Philadelphia, and 1966, Las Vegas.

It reaffirmed its opposition to compulsory coverage of physicians under the Social Security Act, after receiving 11 resolutions opposing coverage and only two favoring the inclusion of physicians.

I have given here only a brief summary of what I feel are the more important subjects considered at this annual meeting of the A.M.A. I am deeply grateful to the Maine

Medical Association for making it possible for me to attend this meeting as your delegate and wish to affirm my confidence in what this most democratic body, the A.M.A., is doing to keep the practice of medicine from becoming a socialistic tool to a federal bureaucracy.

ASA C. ADAMS M.D.

## SPECIAL COMMITTEES 1962-1963

*(Continued from July issue)*

### School Health Committee

Norman E. Dyhrberg, M.D., 323 Main St., Cumberland Mills — Chairman

Margaret S. Smith, M.D., Box 967, Presque Isle

Marion K. Moulton, M.D., West Newfield

### Maine Committee — American Medical Education Foundation

Robert W. Agan, M.D., 144 State St., Portland — Chairman

Charles R. Glassmire, M.D., 58 Deering St., Portland

Paul A. Fichtner, M.D., 6 Pleasant St., Rangeley

### Committee On Conservation Of Vision

Dexter J. Clough, 2nd, M.D., 224 State St., Bangor — Chairman

Howard F. Hill, M.D., 33 College Ave., Waterville

Paul Maier, M.D., 723 Congress St., Portland

Paul E. Floyd, M.D., 2 Middle St., Farmington

Otis B. Tibbetts, M.D., 181 Gamage Ave., Auburn

Ralph A. Goodwin, Jr., M.D., 33 Court St., Auburn

*(To be continued)*

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order of business in the House of Delegates folder as was the report of the Council chairman, the report of the Secretary-Treasurer, and Councilor reports. Reports not submitted, but presented at this meeting, will be sent to the members of the House of Delegates, and pertinent items brought out as this resume of the Stenographic Record proceeds.

### Report of the Executive Director

The report of the Executive Director, Daniel F. Hanley, M.D., covered many subjects of interest, including the Medical School for Maine, the Chiropractic Bill, the Kerr-Mills bill, the Social Security poll, the M.M.A. Committee on Recruitment, Aid and Placement, Maine Medical Education Foundation, and a request from the Attorney General of the State of Maine.

Copies of this report will also go to the members of the House of Delegates but there are two items which I feel should be mentioned here.

First, the Maine Medical Education Foundation. Dr. Hanley stated, "You have contributed to this over a period of two years, now. Your fund now stands at \$41,000, with \$5,000 from the Bingham Fund. . . ."

"This year, only 500 doctors from Maine contributed and this is a smaller number than contributed the first year. This is largely due to misunderstanding on the part of some of the physicians. . . ."

"Again, I want to state that we are not going to pressure anybody into giving. We are going to ask you and ask you repeatedly to take this back to your county societies and explain it to them.

"This year the doctors of Maine, to their own Foundation contributed \$12,500, and from outside sources we have received \$13,500. . . ."

"We sincerely believe it is a good program.

"Last year, we loaned out \$1,400.00 to needy, worthy medical students from Maine.

"This year, we have requests for financial assistance for medical students, amounting to \$8,600.00. We can meet it. And we can meet it next year, but only with your help. . . ."

"Now, I think, personally, that programs like this are the answer to a lot of problems that the doctors face today. Here is a practical public relations program for the people of Maine and the other states in the country to look at."

Second, the letter from the Attorney General concerning the Medical Examiner Law, Chapter 89 of the Revised Statutes of Maine. This law was published in the September, 1961 issue of The Journal of the Maine Medical Association. Special attention is called to that Section of the law concerning death without medical attention. We urge every member to read this law with care.

### Social Security

Dr. Hanley stated that the results of the Social Security poll, authorized at the meeting in April, was 454 in favor of physicians being covered by Social Security and 209 opposed. A resolution that the House of Delegates instruct its Delegate to the A.M.A. to attempt to secure legislation by the House of Delegates of the A.M.A. to implement the inclusion of physicians in the provisions of the Social Security Act was lost. Therefore, our Delegate to A.M.A. is not instructed to take any action on Social Security.

David K. Lovely, M.D., of Portland, read an editorial from the June 1st issue of the Wall Street Journal, "apropos of Social Security in general." It was voted that copies of this editorial be sent to every members of the Society.

*To be continued*

### Hypertension Secondary To Renal Artery Occlusive Disease\*

"Historically, the awareness of renal artery narrowing as a curable cause of hypertension has evolved gradually, highlighted especially by Goldblatt and Poutasse. The increasing use of renal arteriography over the past eight years has yielded a total of 70 patients with renal artery abnormalities out of 110 hypertensive patients examined. . . . Our experience to date indicates that by doing arteriograms in individuals with unexplainable recent onset or exacerbation of hypertension, papilledema, and epigastric bruit, atherosclerosis of the abdominal aorta, or disparity of renal size or function, one is likely to discover the majority of patients with arterial abnormalities." (Reference: Perloff et al., *Circulation*, Volume XXIV, 1961, pages 1286-1304.)

### Announcement

The New Haven Heart Association in cooperation with the Connecticut Heart Association, New England Affiliates of the American Heart Association, and the Yale-New Haven Medical Center will present the M. Grant Blakeslee Memorial Program on "Clinical Problems in Scuba and Skin Diving and Passenger Flying" on September 21, 1962, at Yale University School of Medicine in New Haven, Connecticut. A program will be made available to all New England physicians by August 15, 1962.

Ed. — JACOB B. DANA, M.D.

\*Submitted by the Maine Heart Association, Inc.



DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## Maine Committee On Adoption\*

RUTH L. CROWLEY, LL.B.\*\*

My purpose here today is not to present an exhaustive thesis on the problems in the field of adoptions in Maine. I am neither qualified for such a task nor does time permit such a presentation and still allot an adequate amount of time for our main speaker (E. Kathryn Pennypacker, Chief, Bureau of Social Services, Delaware) who has had much practical and professional experience in this field. Rather it is my hope that with facts and figures I may be able to startle you all to the extent that you will resolve to return to your respective communities and initiate a program of education among your fellow citizens to better acquaint the public with the existing problems and the legislative reforms which may be required to alleviate these problems.

Before we delve into facts and figures, I think each of you will be interested to know some of the background data for the formulation of this Maine Adoption Committee.

Taking a long, hard and scrutinizing look at the adoption situation in Maine is not a new idea. Similar studies have been made sporadically over a period of ten or more years.

In 1952 the Department of Health and Welfare conducted a study of independent adoptions — that is, adoptions arranged and completed without consultation with a private or public agency established for this purpose — and as a result of this study which covered a two and a half year period, made four recommendations, namely:

1. A stronger licensing or placement law that would limit to authorized agencies the adoptive placement of children with non-relatives.
2. A statutory amendment providing that in all independent adoptions involving non-related persons a mandatory study be conducted by the Department of Health and Welfare or by some authorized agency with a report to be made to the court prior to the granting of the adoption.

3. A large-scale educational program aimed at alerting the public to the need for agency participation in non-related adoptions.

4. A statutory amendment to require at least six months' residence for petitioners prior to adopting in a Maine court.

The Department has continued its educational program, but to date, no change in the law has been effected.

In 1953 the Advisory Committee to the Department of Health and Welfare appointed a sub-committee and commissioned its members to study the Department's own adoption program and the overall situation existing in Maine in respect to independent adoptions. This sub-committee recommended statutory amendments requiring at least six months' Maine residence for adoptive parents, and mandatory studies and recommendations by authorized agencies in all non-related adoptions, and providing for parental consent to an adoption to be made *only* before a Judge of Probate (as opposed to the current provision that such parental consent is made before a Justice of the Peace or a Notary Public).

As a result of the recommendations of this sub-committee a bill was introduced into the 97th Legislature in 1955 requiring the six months' Maine residence for adoptive parents. But the fate of this bill was decided in committee where it was reported out unfavorably and thus was not passed.

In 1956 the Research Committee of the Maine Welfare Association undertook a survey of the mandatory study law. At that time thirty-eight states had such a law, and I should interpolate to say that at present at least forty-one states have such legislation in effect. This Research Committee polled the Probate Judges of Maine with the result that five indicated themselves to be favorable, eight were opposed and three offered no opinion. Six of these Judges were willing to refer all non-relative adoptions to the Department of Health and Welfare or to an authorized private agency for study during a trial period to see how effectively it would work.

Last summer at the New England Health Institute

\* A talk before the Maine Adoption Committee, April 25, 1962, Augusta, Maine

\*\* Assistant-Attorney General-Counsel for the Department of Health and Welfare, State House, Augusta, Maine

held at Colby College a workshop on adoptions was scheduled. Panelists were Warren C. Baldwin, M.D., Portland, Mr. Herman Levin, Director of the Portland Child and Family Services, Miss Mary Sullivan, Director of Public Health Nursing in the Department of Health and Welfare, and Mr. Albert F. Hanwell, Director of Child Welfare in the same Department. It is interesting to note that all four of these able panelists are members of this new Maine Adoption Committee here assembled. The outcome of this group discussion was a recommendation for the formation of a multi-disciplinary committee to work toward more adequate legislation in the field of adoptions.

In September 1961, a representative group from child placing private and public agencies started a series of conferences to identify the problems and to work toward the formation of the recommended multi-disciplinary committee. It was my privilege to be a part of this group, invited by them to join them in order to render my interpretations of the existing laws. The Maine law was carefully studied and its many serious weaknesses were revealed. Finally, this group carefully selected the members to be invited to participate in the Maine Adoption Committee. Selection of a chairman was its first task, and may I say that I feel personally that this committee has been most fortunate in obtaining the services of Mr. Peabody (Arthur A. Peabody, Attorney at Law, 97 A Exchange Street, Portland, Maine) who already has exhibited very capable leadership and a keen interest in the problems with which we are faced.

So now that you know how this committee came into being, let us briefly look at the basic statutory provisions as they exist at present. The entire adoption law is contained in eight sections of Chapter 158 of the Public Laws of 1954. Both residents and non-residents may adopt in this State. Provision for simple consent of the natural parents is included, as I have previously noted, or for consent of the Department of Health and Welfare in the case of children committed by court order to its custody. There is also a provision for a parent or parents to surrender a child for adoption to an authorized agency, with such surrender to be signed before a Judge of Probate Court who has the responsibility of explaining the effect of such a surrender. In 1959 new legislation was enacted which made it possible for the Judge of Probate to enlist the services of the Department of Health and Welfare in making a study of circumstances in an adoption, but this is wholly optional and not mandatory. It should be added that some of our Probate Judges use this service quite freely while others disregard it almost entirely. Provisions in our statutes make it optional, but not mandatory, for the Judge to require a one-year trial period in an adoptive home before the adoption is granted. As a matter of fact, this option is rarely invoked by our Judges in independent adoptions. The balance of the adoption statute provides that the records shall be confidential, and provides that an adopted child shall have rights of

inheritance from both his natural and his adoptive parents. Obscured in the licensing law is a very ambiguous provision which says that no person, firm, corporation or association shall engage in, or assist in conducting a business or placing out or finding homes or otherwise disposing of children under sixteen years of age unless licensed by the Department of Health and Welfare. This law, with its provision for a fine of an amount up to \$500 and/or imprisonment of not more than eleven months, has not been tested but it is believed to have so many loopholes that conviction under it would be difficult in the cases of persons placing or assisting in placing children for adoption without license from the Department.

Because of the obscurity and probable ineffectiveness of this law, we have a widespread practice in this state of independent adoptions in which arrangements for placement are made by professional and non-professional persons with little or no concern for the rights of the child, of the adoptive parents and of the natural parents.

As I look about, I can see that for many of you a question is immediately posed: What is the reason for believing that adoptive placements made without a careful study of all factors involved are risky and can result in grave problems from both an emotional and a legal point of view? I am sure that none of us who are working intimately with this problem either as trained social workers or as legal consultants, would arbitrarily set ourselves up as authorities to answer this query which is a very normal and natural one for you to have. However, armed with statistics and with tragic examples of poor adoptive placements which have been brought to our attention over a period of years, we feel justified in saying that much unhappiness may result from poor placements — unhappiness and grief for the adoptive parents, for the children involved, and frequently for the natural parent or parents.

A study undertaken at Yale University included a sampling of one hundred agency placements and one hundred independent placements. The findings indicate that 86% of the agency placements were successful while only 46% of the independent placements were successful. These figures are consistent with similar studies made elsewhere. To be sure, we cannot guarantee that agency placements will completely eliminate unsuccessful adoptions, for there is always present the possibility of human error in judgment. But surely these figures quite vividly show that there is less risk involved in an agency placement than in an independent one.

In the Department of Health and Welfare, as in private agencies, we have long been aware that a limited black market and an extensive gray market in adoptions exist in Maine. Perhaps some de-identified cases will best prove this point.

An unwed mother from New York State was taken to Boston where she delivered her baby. In both New York and Massachusetts the law would not permit adop-

*Continued on Page 210*

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DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page 208*

tion of this child independently, so the mother and infant were brought to Maine where previous arrangements had been made for an adoption to be effected for a New York couple. The whole process consumed less than a day's time so that by night of the day when mother and child came to Maine she had divested herself of all parental rights and given her child to a couple who were total strangers to her.

In another case a widowed mother of several children had a baby out of wedlock. On the day following the baby's birth two doctors approached this mother in the hospital and told her how bad it would be for her other children if she were to take this new baby home and offered to arrange for its adoption by "friends." Still undecided, the mother took her baby home and agreed to get in touch with the doctors when she had reached a decision. Within hours after her arrival home, she was contacted by an attorney who urged her to come to his office and sign a consent for adoption. Under all this pressure, she did sign a consent. A day or two later she learned that the adoptive parents were of a different religious faith from hers and that the child was being taken outside the continental United States to live. She returned to the attorney's office and said she had changed her mind about the adoption and wanted her baby back. That night the child was flown to Puerto Rico without the adoption being completed. The Puerto Rican authorities have been advised of this mother's revocation of consent and the report several months after this episode is that no adoption petition has been filed in Puerto Rico. The fate of this child is at present unknown.

In another instance a childless couple inquired from a doctor about the possibility of obtaining a child for adoption. With the doctor the couple went to another town, and brought back a newborn infant, for whom the couple paid the doctor \$200. An adoption was effected, and later the adoptive mother developed severe emotional problems involving compulsive check forgery and suicidal tendencies, and the father became a chronic alcoholic.

An Air Force couple were allowed to adopt an illegitimate child without benefit of a study. After the completion of the adoption, it was learned that the adoptive father was, at the time of the adoption, a patient completely disoriented in the psychiatric ward of the base

hospital. The adoptive mother, discovered to be a promiscuous woman, left the state with the child after about a month and as far as is known is living with another man without benefit of wedlock.

Other cases involve adoptions by mentally deficient adopting parents, by chronic alcoholics, by a mother so crippled that she is unable to hold or care for her child, and by adopting fathers who have sexually molested their very young adopted daughters.

Do these facts shock you? I hope so, for that was my purpose in selecting such appalling cases.

Our problem in Maine is accentuated by the fact that we have one of the highest illegitimacy rates of any of the states. Unwed mothers fall easy prey to unscrupulous persons interested in conducting a black or gray market in adoptions. However, we do not wish to point a finger at any profession. Physicians, lawyers, ministers, and other professional and non-professional persons, many acting in good faith with good intentions and with no idea of violating or evading the law, help to place children in adoptive homes.

In the past ten years over two thousand children have been placed in unknown situations with non-relatives without benefit of a professional evaluation of the facts. Nearly five thousand more have been placed with relatives with no professional service to insure a stable and secure placement.

Each year has seen the number of independent adoptions, particularly those to out-of-state adopting parents, on the steady increase. It would seem that we must look searchingly at the present statutory provisions to see what can be done to alter this threat to the welfare of an increasingly large segment of our children. Yours — or more correctly, I should say ours since I am a member of this committee — ours is the responsibility to study the problem, to recommend changes to improve the situation, to educate our fellow citizens to the existing problems and their alleviation, and perhaps most difficult of all to convince the legislators of the need for statutory amendments to help solve this problem. If by our work, we save even a few children from the fate of poor adoptive placements, then we can truly feel that our work has not been in vain. I ask you — are you ready to accept this challenge to protect the welfare of adopted children in Maine? I certainly hope so, for this is our task as members of the Maine Adoption Committee.

1962 SCIENTIFIC SESSION

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MONDAY, OCTOBER 22

MORNING SESSION

9:00 A.M.—12:30 P.M.

PROLOGUE

Chairman:

Dr. Kenneth M. Endicott  
National Cancer Institute  
Bethesda, Maryland

*Milestones in Cancer  
Control—The Past 25 Years*

Dr. John R. Heller  
Memorial Sloan-Kettering  
Cancer Center  
New York, New York

*The Epidemiology of Cancer  
—Scope and Trends*

Dr. Lester Breslow  
State Department of  
Public Health  
Berkeley, California

CAUSATION OF CANCER

Chairman:

Dr. Sidney Farber  
The Children's Cancer  
Research Foundation  
Boston, Massachusetts

*Chemical Carcinogenesis*

Dr. Albert Tannenbaum  
Michael Reese Hospital  
Chicago, Illinois

*Physical Agents as Causative  
Factors*

Dr. Austin M. Brues  
Argonne National  
Laboratory  
Argonne, Illinois

*Viruses and Cancer*

Dr. Wendell M. Stanley  
University of California  
Berkeley, California

*Tobacco and Cancer*

Dr. Ernest L. Wynder  
Sloan-Kettering Institute  
for Cancer Research  
New York, New York

*Tobacco and Cancer—Acting  
on the Evidence*

Sir Robert Platt  
Royal College of  
Physicians of London  
London, England

AFTERNOON SESSION

2:00 P.M.—5:00 P.M.

BIOLOGICAL ASPECTS  
OF CANCER

Chairman:

Dr. Shields Warren  
Harvard Medical School  
Boston, Massachusetts

*The Chemical Basis of  
Genetics as Related to  
Cancer*

Dr. Vincent Allfrey  
The Rockefeller Institute  
New York, New York

*Cancer Cells: Enzyme  
Localization and  
Ultrastructure*

Dr. Alex B. Novikoff  
Albert Einstein College of  
Medicine  
New York, New York

*Cellular Differentiation  
and Neoplasms*

Dr. Thomas J. King  
The Institute for Cancer  
Research  
Philadelphia, Pennsylvania

*Susceptibility and Resistance  
to Cancer*

Dr. Chester M. Southam  
Sloan-Kettering Institute  
for Cancer Research  
New York, New York

*Cancer Cell Spread*

Dr. George E. Moore  
Roswell Park Memorial  
Institute  
Buffalo, New York

TUESDAY, OCTOBER 23

MORNING SESSION

9:00 A.M.—12:30 P.M.

DETECTION, DIAGNOSIS  
AND TREATMENT OF  
CANCER

Chairman:

Dr. Eugene P. Pendergrass  
University of Pennsylvania  
Philadelphia, Pennsylvania

*Problems and Progress  
in Cancer Detection*

Dr. Emerson Day  
Memorial Hospital for  
Cancer and Allied Diseases  
New York, New York

*Cytologic Diagnosis of  
Cancer—Its Present and  
Future*

Dr. Leopold G. Koss  
Memorial Hospital for  
Cancer and Allied Diseases  
New York, New York

*Radiology in Cancer  
Detection and Diagnosis*

Dr. Wendell G. Scott  
Washington University  
School of Medicine  
St. Louis, Missouri

*The Potential of New  
Techniques Including  
Automation in Cancer  
Detection*

Dr. George Z. Williams  
U.S. Department of  
Health, Education &  
Welfare  
Bethesda, Maryland

*Progress and Prospects  
in Cancer Staging*

Dr. Murray M. Copeland  
M.D. Anderson Hospital  
& Tumor Institute  
Houston, Texas

*Surgery in the Treatment of  
Cancer—Present Status*

Dr. Warren H. Cole  
University of Illinois  
College of Medicine  
Chicago, Illinois

*Radiotherapy of Cancer—  
Present Status*

Dr. Richard H. Chamberlain  
University of Pennsylvania  
Philadelphia, Pennsylvania

AFTERNOON SESSION

2:00 P.M.—4:00 P.M.

DETECTION, DIAGNOSIS  
AND TREATMENT OF  
CANCER (cont.)

Chairman:

Dr. Stuart M. Sessoms  
National Cancer Institute  
Bethesda, Maryland

*Hormone Therapy of Cancer*

Dr. Charles Huggins  
University of Chicago  
Chicago, Illinois

*Formulation and Evaluation  
of Chemotherapeutic Agents*

Dr. Howard E. Skipper  
Southern Research  
Institute  
Birmingham, Alabama

*Systemic Chemotherapy*

Dr. C. Gordon Zubrod  
National Cancer Institute  
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*Adjuvant Chemotherapy of  
Cancer*

Dr. I. S. Ravdin  
University of Pennsylvania  
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# The Journal of the Maine Medical Association

Volume Fifty-Three

Brunswick, Maine, September, 1962

No. 9

## President's Address\*

JAMES A. MACDOUGALL, M.D.\*\*

The Constitution and By-laws of the Maine Medical Association state that one of the duties of the president is that he shall deliver an address at the annual session. Coming from one who for many years has enjoyed the kind and thoughtful exemptions accorded to senior members, I feel that this address should take the form of a valedictory rather than any attempt at oratorical exhortation. With this in mind, and without presuming to borrow greatness from the great, I am reminded of the opening paragraph of one of Sir William Osler's inimitable addresses "After Twenty-five Years," let me quote:

"From two points of view alone, have we a wide and satisfactory view of life — one, as amid the glorious tints of the early morn ere the dew of youth has been brushed off, we stand at the foot of the hill eager for the journey; the other wider, perhaps less satisfactory, as we gaze from the summit at the lengthening shadows cast by the setting sun. From no point in the ascent have we the same broad outlook, for the steep and broken pathway affords few halting places with an unobstructive view. You remember in the ascent of the mountain of Purgatory, Dante, after a difficult climb, reached a high terrace encircling the hill and sitting down turned to the east, remarking to his conductor, 'All men are delighted to look back'."

Thus it is on this occasion, after forty-two years, with the dew of youth long since brushed off and in the ocidental phase of life's trajectory, I am delighted to look back and pass in review some of the achievements which adorn that period.

In assessing the import of those years and their impact on the healing arts, I think we have lived through one of the most exciting and progressive eras in the history of medicine. To enumerate all the advances achieved in this period would fill a volume, but to many of you in practice today, what is commonplace daily routine, was unknown to us when I first assumed the duties of attempting to heal the sick.

I happened to open one of my old note books a short time ago and to read notes taken at lectures on diabetes mellitus. How dismal was the prognosis and how futile the treatment — all to be so happily transformed in the following year by the advent of insulin. Next came the use of liver extract in the treatment of pernicious anaemia, before which, the outlook was a slow and fatal decline with occasional remissions. Sulfa therapy then appeared to take its place in the combat with the "captain of the men of death" — pneumonia. I remember well the first patient I treated for pneumonia with Sulfapyridine. She was feeling well in four days from the onset of symptoms and, because recovery was so rapid, she became convinced that my diagnosis was wrong, that I didn't know what I was doing and refused to pay me for my services; nor was the pharmacist paid for the sulfa tablets, as she considered his charges exorbitant. Sulfa was followed by penicillin and the antibiotics, about the use of which much could be said and, I fear, much will be said as a result of the insidious resurgence of resistant organisms. To the above should be added the triumphs over tuberculosis, the obliteration of the scourge of typhoid fever and the spectacular reduction in infant mortality as a result of hygienic food handling and immunization programs, all of which resulted in greater life expectancy — and lives not harried by the fear of impending epidemics.

\*Presented at the 1962 annual session of the Maine Medical Association.

\*\*President, Maine Medical Association, 1961-1962.

This is indeed a roseate picture of medicine and at that but poorly limned, but where, as a profession, do we stand today in prestige and public esteem in comparison with the doctor of half a century ago? Where will you find today, the loyalty and undeviating faithfulness to the physician which enriched, if nothing else did, the labors of the doctor of fifty years ago? Only among those of the older generation, certainly not in these of the assembly-line age.

Why is this so? Probably we can search and discover some of the reasons in our own deportment.

Science is fast displacing art in the practice of medicine and we are fast becoming devotees of science to the exclusion of the "savoir-faire" of art. Pushed to its logical conclusion, it is not difficult to envision the day or decade when diagnosis will be determined by subjecting the patient to some blood-letting and abstraction of other body fluids, all to be introduced to a computing machine, or some variation thereof, the verdict of which shall be interpreted as the sum total of the patient's indisposition. This would be the ne plus ultra in the dehumanizing process of science in medicine and would work to the detriment of all that is ideal in the art of medicine.

"The proper study of mankind is man," is as true today as when it was first enunciated and man happens to be composed of two very distinct entities; in health these are in perfect accord; in disease that accord is disrupted and whilst the computing machine may divulge some pathological aberration of the corporeal entity, the psychic, the spiritual entity, is disregarded only to be reached by the humanizing touch of the art of medicine. Specialization, with its concentration on the part, and its brief acquaintance with the patient, is contributing much to this apathy towards the profession. Not that I would have fewer specialists, but more general practitioners of whom, and again I quote Sir William Osler, who spoke as follows: "They form the very sinews of the profession, generous-hearted men with well-balanced, cool heads, not scientific always, but learned in the wisdom, not of the laboratories, but of the sick room."

It is he who sees the patient in his home environment who is conversant with all the externals which might contribute to, or militate against, his recovery, and, therefore, best qualified to prescribe adequate care and treatment.

And now we have taken to the hustings. For many years we proclaimed that the vote was the individual's personal prerogative and that he exercise this privilege as a citizen, not as a member of any class, profession or organization. This ideal apparently no longer holds, and, as a result, we find ourselves headed for the miasmatic swamp of party politics. What we formerly eyed with

suspicion in other groups, we now vigorously practice ourselves. It is told that after the woman's suffrage bill became law, two U. S. senators were discussing the novel situation when one proposed a toast to the ladies. Raising his glass he said: "Here's to women. Once our superiors, now our equals." This is quite apropos to us at the present time. We have placed ourselves on the same level as any trade or commercial organization and can expect consideration from the public accordingly. It has been well said that "he who keenly engages in political controversy must not only encounter the vulgar abuse which he may justly condemn, but the altered eye of friends whose regard has been chilled" — and we need friends.

In view of our present disorientation with regard to our status as a profession, I was not surprised to find a columnist in a recent issue closing his disquisition on this problem by saying, "But I wonder if they are going to regain as a class the high degree of respect that they have always enjoyed; and if I were a doctor, I would regard the slowly changing public attitude toward my profession with some concern."

The age of the rugged individual has gone by. It is a far cry from the first rugged individual, the cave man, whose law was the club and claw, to our present mode of social existence. Over the last thirty years we have undergone a social revolution and this will go on as relentlessly as the tides and as long as man strives for better relations with his fellow man. This is no longer a local problem, but a universal one by virtue of easier and more rapid inter-orbital communications.

In conclusion, let me state that I feel that ours is only an infinitesimal part of the turmoil that today disturbs the world. I hope and pray that common sense and good judgment may prevail in the solution of our problem and that we may remain ever worthy of that beautiful tribute paid to our profession by Robert Louis Stevenson when he wrote: "There are men and classes of men that stand above the common herd; the soldier, the sailor and the shepherd not infrequently; the artist rarely; the physician almost as a rule. He is the flower of our civilization; and when that stage of man is done with and only to be marveled at in history, he will be thought to have shared as little as any in the defects of the period and most notably exhibited the virtues of the race. Generosity, he has such as is possible to those who practice an art; never to those who drive a trade; discretion, tested in a hundred secrets; tact, tried in a thousand embarrassments; and what is more important, Heracleian cheerfulness and courage, so that he brings air and cheer into the sick room and, often enough, though not as often as he wishes, brings healing."

# Electrocardiographic Interpretations In Late Prenatal And Early Neonatal Life\*

EDWARD M. SOUTHERN, M.D.

This report deals with the electrocardiographic reflection of the haemodynamic changes that occur in the transition from late prenatal to early neonatal life. The dependence of the fetus on feto-maternal gas exchanges during intrauterine life is suddenly and dramatically cut off at birth, so that survival in an extra-uterine environment rests upon the establishment and efficiency of adequate pulmonary function.

The adjustments of the fetal circulation at birth in the human include the occlusion of the placental circulation by constriction of the umbilical arteries, the functional closure of the ductus venosus and the changes of blood flow via the foramen ovale and the ductus arteriosus. An adequate stimulation for the functional closure of the ductus arteriosus appears to be an increased oxygen tension of the blood.<sup>1</sup> The blood pressure in the ventricles contributes to the neonatal adjustment of systemic circulation.

Apart from the difficulties of technique in measurements, there are many factors that have to be considered in any attempt at an interpretive hypothesis of prenatal cardiac complexes. The effects of uterine contractions in labor upon placental blood flow are reflected by haemodynamic alterations in the fetal heart associated with changes in the volume of blood flow in the placental intervillous space; the intramyometrial pressure changes in labor may decrease the arterial inflow and venous outflow in the intervillous space. Other factors include, (1) lack of pulmonary ventilation and minimal pulmonary blood flow "in utero," (2) the patency of the ductus arteriosus (3) the possibility of some flow through the ductus arteriosus from the aorta to the pulmonary trunk — this has been demonstrated in fetal lambs by cine-angiography.<sup>2</sup>

Many of these variables are not yet susceptible to accurate measurement in the human and, therefore, only a tentative study of changes in fetal cardiac energy is possible. Improvements in the technique of prenatal and neonatal electrocardiographic methods have, however, pointed a way to an attempt to study the problem in terms of electrocardiographic data during and after the birth process.

## MATERIAL AND METHOD

The patterns discussed in this study were recorded

from 21 patients in a combined analysis of fetal prenatal and early neonatal electrocardiograms. The neonatal electrocardiograms were obtained directly from conventional and precordial unipolar leads and recorded in a range of 6 minutes to 15 minutes after birth. The prenatal recordings were obtained from maternal abdominal leads in late labor by techniques previously outlined.<sup>3</sup> Simultaneous recordings of variations in uterine contractility by amniotic fluid pressure measurements were obtained by an intra-amniotic catheter and transmitted via a strain-gage manometer (Sanborn) recording through a second channel of the recording unit. For prenatal recording deflections were standardized at 25 cms per millivolt input and paper speed was at 25 mm per second.

## RESULTS

A comparison of the electrocardiographic characteristics was undertaken in the same infants at 6-15 minutes before the end of the first stage of labor and then at 6-15 minutes after birth. The birth weights varied from 4200 to 3250 Grams. Congenital cardiac abnormalities were excluded, as far as possible, by clinical observation and electrocardiographic recordings. No sedation was used for the infants. The results are illustrated in Table I.

The prenatal electrocardiograms, with a variation of fetal heart rate of 101-150 beats per minute ( $S.D. \pm 3.2$ ) from 6-15 minutes before birth and a frequency of intervals between uterine contractions of 0.45 seconds to 2 minutes were contrasted to neonatal recordings from 6-15 minutes of extra-uterine life with heart rates of 120-162 beats per minute ( $S.D. \pm 5.2$ ).

1. *P.R. Interval* Before birth the variation was 0.06-0.13 sec: This, presumably, corresponds with a shortening of P-R at relatively rapid heart rates because of decreases stroke volume and the small diameters of conduction tissue. A similar decrease of P-R with heart rate is found in many smaller animals with rapid rates.<sup>4</sup> In the immediate neonatal phase the interval was 0.05-0.19 sec, so this seems to indicate no appreciable alteration in atrio-ventricular conduction at birth. The P-R duration decreases slightly in the course of the first week of life.<sup>5</sup>

2. *R/S Ratio* In this small series, the R/S ratio in the prenatal heart recordings was one or greater in 19 cases out of 21 and in the neonatal group R/S ratios in right precordial leads were greater than one in 17

\*Project supported by research grants from The Maine Heart Association and The Raphael Fund at the Thayer Hospital and Colby College, Waterville, Maine.

TABLE I

PRENATAL								POSTNATAL				
Case	Sex	Heart Rate	Mins. Before Birth	Uterine Contractions Per Min.	P-R	R/S Ratio Greater Than 1	T Wave	Heart Rate	Age In Minutes	R/S Ratio Greater Than 1	T Wave	P-R
1	M	116	13	2	0.09	*	U	134	6	*	U	0.09
2	M	136	10	2	0.10	*	U	142	6		U	0.12
3	M	130	12	1	0.05	*	U	162	8	*	U	0.15
4	F	140	12	1	0.07	*	Is	150	12	*	U	0.08
5	F	146	10	1.5	0.12	*	U	136	10	*	U	0.09
6	M	126	15	0.45	0.11	*	U	150	6	*	U	0.10
7	F	130	6	.1	0.06		In	146	12	*	In	0.12
8	M	102	7	0.50	0.03	*	U	138	15	*	U	0.09
9	F	117	10	1	0.09	*	U	128	10		In	0.08
10	F	130	12	2	0.07	*	Is	136	10		U	0.11
11	F	140	15	2	0.12	*	In	148	12		U	0.15
12	M	150	12	2	0.13	*	In	152	6	*	In	0.19
13	M	146	17	1	0.08	*	U	142	8	*	U	0.08
14	F	104	15	1	0.06	*	U	125	7	*	U	0.09
15	M	101	5	0.45	0.08	*	U	160	8	*	U	0.07
16	F	140	10	1	0.07		U	130	10	*	U	0.05
17	F	138	14	2	0.10	*	U	138	15	*	U	0.09
18	M	142	15	2	0.12	*	U	150	7	*	In	0.17
19	M	150	10	2	0.06	*	In	148	10	*	U	0.12
20	M	120	15	1.5	0.08	*	Is	130	12	*	U	0.13
21	F	119	8	0.70	0.07	*	U	120	10	*	In	0.09

cases of the 21. The characteristic right axis deviation appears to be present both before and after birth. There was, however, some variation in the remaining 4 cases in neonates, with a more adult-type pattern, certainly not characteristic of so-called "neonatal ventricular hypertrophy."

3. *T Waves* Upright T waves were discernable in 14 prenatal recordings and 16 neonatal patterns (right precordial leads). The positivity of T waves during and shortly after the birth process appears of uncertain significance in this study. It does not appear to reflect any pathological right ventricular hypertrophy or hypertension. The diagnosis of pathological ventricular hypertrophy cannot rest upon the slim evidence of scattered T wave changes in conjunction with right ventricular preponderance.

Arrhythmia was only present in 2 cases in this study before birth and in one case it persisted into the neonatal phase. The character was of ventricular premature systoles.

#### DISCUSSION

It has been claimed, that in the fetus the left ventricular output exceeds that of the right ventricle. Keen<sup>6</sup> has made observations on the weight of the right ventricular mass and its parts in the post-natal infant

heart. He also measured the thickness of the ventricular walls in order to compare the changes in weight. In 19 specimens (at full term), the mean right ventricular thickness (measured from the infundibulo-ventricular crest — crista supraventricularis) was 6.1 mms and that of the left ventricle, between the anterior and posterior groups of papillary muscles was 5.2 mms. At age 4-6 months the mean RV thickness was 3.8 mms as compared to 8.0 mms in the left ventricular wall. These observations suggest that there is atrophy of the RV wall in normal newborns. In terms of functional capacity, the increase in the volume in the right ventricle would be  $(1.5/1.0)^{3/2}$  equal to 1.8 and that of the left ventricle  $(1.9/1.5)^{3/2}$  or equal to 1.4. In prenatal life, the fetal ventricles act against the same peripheral resistance and thus a lesser capacity implies a lesser output. The inference in these findings is that the functional capacity of the right ventricle before birth is less than that of the left ventricle. Immediate neonatal alterations of mean heart volume, calculated by area measurements and determined by roentgenological techniques, have been reported by Lind,<sup>7</sup> and by Kjellberg, Ruhe and Zetterstrom.<sup>8</sup> These observations, in the first 5 days following birth, showed a progressive change in mean heart volume of 48ml, 40ml, 38ml, 35ml and 30ml respectively in these five

FIG. 1-A

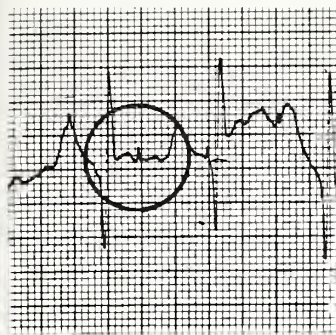


FIG. 1-B

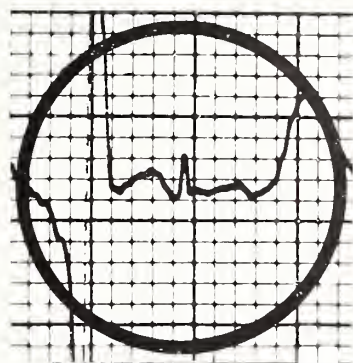


Fig. 1-A. Maternal and fetal prenatal complexes 6 minutes before birth.

Fig. 1-B. Enlarged fetal complex showing P-R interval in late labor.

FIG. 3-A

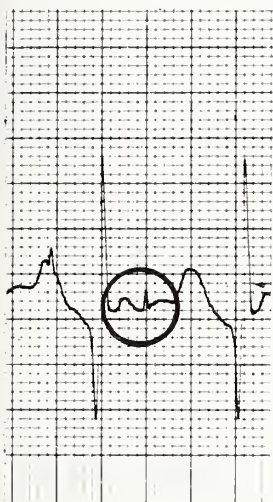


FIG. 3-B

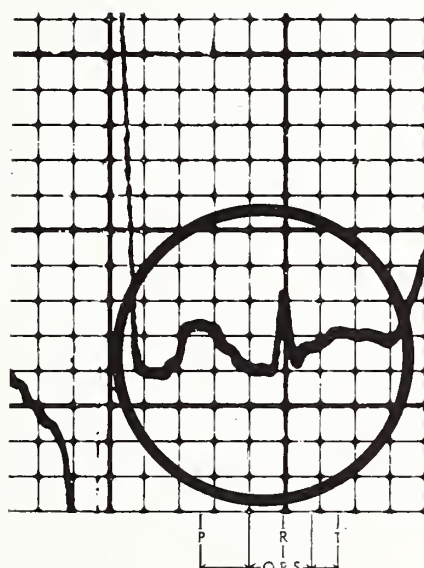


Fig. 3-A. Maternal and fetal prenatal complexes 6 minutes before birth.

Fig. 3-B. Enlargement of fetal complex from above.

FIG. 4-A

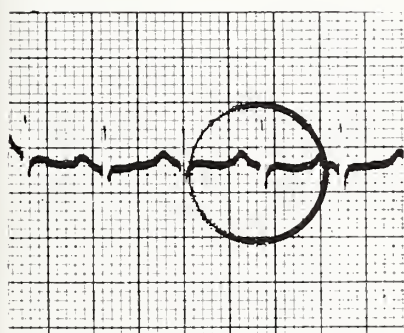


FIG. 4-B

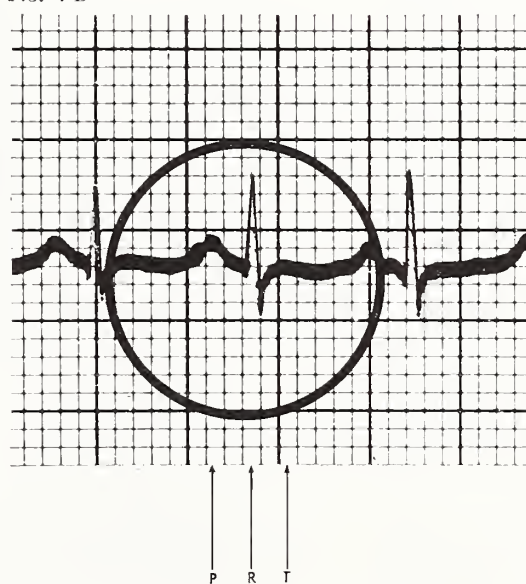


Fig. 4-A. Neonatal complexes (4 minutes after birth) from the same baby as Fig. 3.

Fig. 4-B. Enlargement of neonatal complexes from above case.

FIG. 2-A

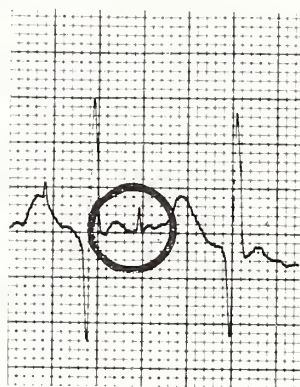


FIG. 2-B

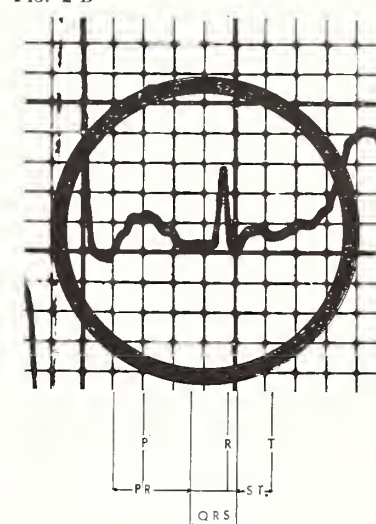


Fig. 2-A. Maternal and fetal prenatal complexes 4 minutes before birth.

Fig. 2-B. Enlarged fetal complex showing timing of components in late labor.

days. The early change is thought to be a reflection of increased peripheral resistance.

It has been alleged that the characteristic feature of the prenatal and early neonatal electrocardiogram is the evidence of right ventricular preponderance.<sup>9,10</sup> The significance of the "upright" nature of the prenatal complexes and of right axis deviation in precordial leads in the newborn, has not as yet been fully clarified. Recent data<sup>11</sup> shows variations from the classical examples of newborn recordings. In some subjects, the R/S ratios are less than one in right precordial leads and greater than one in left precordial leads. It has been suggested that ventricular hypertrophy may not be the dominant factor and that heart position, rotation and the relatively thin anterior chest wall of the newborn may influence the picture. Rosen and Gardberg,<sup>12</sup> in a combined study of newborn electro-

cardiograms and vectorcardiograms conclude that, "in the vectorcardiogram, the horizontal projection of the QRS loop would not be expected to be inscribed in a clockwise manner unless the right ventricle was thicker than the left." Since they did not find this to be true, even during the first few months of life, they believe that a distortion inherent in the cube system of vectorcardiography exaggerates the right ventricular effects to produce the clockwise inscribed loops seen in the records made with this method. The present study tends to indicate that whatever the factors are that produce right axis deviation in the newborn, they are present before birth to a large extent and at a time when the peripheral resistance in the ventricles is approximately even.

The second important feature is the positivity of T waves in the right precordial leads in the first 24 hours

of extra-uterine life with progressive inversion of this deflection during the subsequent 3 to 4 days. In the immediate prenatal tracing, the T wave showed upright deflection in normal infants (with  $47.5 \pm 5.1$  per cent value for oxygen saturation from the umbilical artery at birth), whereas with clinical fetal hypoxia ( $17.1 \pm 4.1$  per cent oxygen saturation at birth) the T waves were isoelectric or inverted in 35 per cent.<sup>3</sup> Gros et al,<sup>13</sup> showed that, "the T wave is upright or diphasic in V4R, V1 and V2 and inverted in V5, and V6 for the first 24 hours of life: it gradually alters so that by the fourth day it is inverted in VR, V1, and V2 and upright in V5, and V6 thereafter." This finding is borne out by Kessel<sup>14</sup> who studied normal newborns in the first 24 hours of life and showed similar T wave changes. The meaning of these T wave changes is not fully understood, as the T wave is more likely to be the "area of unexplained changes in the neonatal period, than any other part of the complex."<sup>15</sup> It has been claimed that the positivity of neonatal T wave changes are a primary deflection — that is to say, that they do not depend on association with changes in QRS complex. Zeigler,<sup>16</sup> explains that a direct relationship between the occurrence of T wave positivity in right precordial leads and the degree of elevation of mean right ventricular pressure exists as measured by cardiac catheterization. He shows that the incidence of positive T waves is zero when mean RV pressure is normal, and that there is an increasing incidence of positive T waves with increasing mean RV pressure and a final decrease of positivity with extreme RV hypertension. The suggestion is that the progressive inversion of T waves may signify reduced pulmonary artery and RV hypertension secondary to the assumption of progressive normal lung development and function. In the experimental animal (rabbit), photographic sphygmomanometric recordings show approximately equal pressures (21 mm Hg and 20 mm Hg) respectively in the fetal right and left ventricle,<sup>17</sup> this data, however, may have little or no application to the conditions in the human. Information from cardiac catheterization in neonates is scanty. Rowe and James,<sup>18</sup> report pulmonary systolic arterial pressures between 40 and 60 mm Hg and systemic pressures between 55 and 85 mm Hg in infants from the second day to the sixth day of life. Thereafter, the pulmonary pressures average 30 mm Hg or less. The possibility of a left to right shunt between pulmonary artery and aorta in the human fetal state may well influence prenatal and early neonatal electrocardiographic recordings.

#### SUMMARY

1. Patterns of late prenatal and early neonatal electrocardiographic recordings in the same infants have been evaluated in 21 cases.

2. P-R intervals before birth ranged between 0.06-0.13 sec and after birth 0.05-0.19 sec and did not seem

to indicate any significant change in atrio-ventricular conduction.

3. R/S ratios in the prenatal heart recordings was one or greater in 19 cases out of 21. In the immediate neonatal phase there was a persistence of right axis deviation in 17 out of 21 cases.

4. The positivity of upright T wave changes does not appear to reflect any pathological ventricular hypertrophy or hypertension.

5. The significance of two major features of neonatal electrocardiograms — right ventricular preponderance and T wave changes — is discussed and compared with the prenatal condition.

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# Modified Electro Therapy Versus Anti-Depressant Drugs

FRANK S. BROGGI, M.D.\*

The value of Electro Shock Therapy in dealing with the endogenous depressions has been so firmly established over the past 15 to 20 years that it should be unnecessary to bring this fact to the attention of the reader. Kalinowsky<sup>1</sup> reporting in 1946, reported failures in affective disorders to amount to no more than 4% in Bennett's and 5% in Osgood's material, unfavorable prognostic features appeared to include various neurotic features. In our series of over 11,000 private treatments, we have felt that the results were practically specific, and that if favorable results from the use of this treatment did not occur, the validity of the diagnosis was questioned. (About 2,000 unmodified treatments were given by this author in another general hospital.)

Although the use of this therapy greatly encouraged those psychiatrists who employed it in the treatment of Manic Depressive Psychosis in cases which previously were only given sedation and custodial care, there were certain definite draw-backs and hazards which prevented its use routinely in all cases of Manic Depressive Psychosis. Elderly patients with cardio-vascular disease and those patients who had made suicidal attempts and suffered from fractures etc. were considered too poor a risk to be candidates for treatment. In the Cerletti-Bini standard Electro Shock treatment, the patient underwent a severe convulsive seizure, which frequently resulted in compression fractures of the dorsal vertebrae, fractures of the long bones, dislocations, anoxia and other annoying complications which prevented further use of this procedure. In every treatment, the patient had some degree of hypoxia and cyanosis which resulted in cardio-vascular strain. Although the cardiac deaths from standard Electro Shock Therapy were relatively rare, (.06%) the instance of vertebral compression fractures from D3 to D6 and fractures of the long bone occurred in some series up to about 28% of the cases.<sup>2</sup> Complications occurred frequently enough to dampen the enthusiasm of many psychiatrists from using this type of therapy.

Since August 1949, standard Electro Shock Therapy has not been used at the Maine Medical Center. Instead, all Electro Shock treatments have been modified by the Department of Psychiatry cooperating with the Department of Anesthesiology. A paper discussing these modifications in detail, was published on September

29, 1955.<sup>3</sup> When this paper was written, because of the fact that the patients no longer underwent any convulsive seizure, and because the term Electro Shock Therapy was frightening to many patients, the authors coined and used the term Modified Electro Therapy after that date.

An excellent paper on this subject was published by Adren & Dick, "The Therapeutic Team Approach to Modified Electro Shock Therapy."<sup>4</sup> In fact, the importance of the team approach is so essential that this author is reluctant to give treatment in those hospitals where there is not a trained team and where they are unfamiliar with the handling of this type of patient.

Ardis,<sup>5</sup> an English writer, states, "We consider that unmodified Electro Shock Therapy is outmoded at the present time. The team approach that we have developed is at least one method of making Modified Electro Shock Therapy available for all patients." At the Maine Medical Center, we would not allow anyone to give standard or unmodified Electro Shock Therapy to any patient as we consider the Modified Technique to be so superior.

Different authors have written concerning the dangers of the Modified Technique. Although one prominent shock therapist refused to use Sodium Pentathol,<sup>®</sup> we do not consider this to be a dangerous drug in the hands of a qualified anesthesiologist. It is quite possible that some of the complications which occurred with this technique have been due to the fact that qualified anesthesiologists were not available. In this author's opinion, Sodium Pentathol is no more dangerous when used with patients suffering from mental illnesses than it is with other types of patients. Many thousands of Sodium Pentathol inductions are administered daily throughout the country, for tooth extractions, inductions, narco-synthesis, and other minor surgical procedures, and there are no reports to the effect that it is a potentially dangerous drug. This author believes it is very effective in the use of Modified Electro Therapy for three specific reasons. First, many patients who have a fear of treatment willingly submit to being put to sleep with Pentathol as a considerable number of them have been treated with this medication previously. Second, if Succinyl-Choline<sup>®</sup> were administered to a patient completely conscious it would be a very unpleasant experience if a patient felt completely paralyzed and unable to breathe. Third, Sodium Pentathol helps to prevent and is the treatment for the post-convulsive ex-

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citements which are seen occasionally following the administration of Modified Electro Therapy. Succinyl-Choline or "Anectine"® is a short acting drug which has to be carefully supervised by the anesthesiologist. In rare instances, some patients react adversely to the drug, i.e., they get a prolonged period of apnea. However, as long as a clear air way is established, and the patient is supported with artificial respiration, the patient is in absolutely no danger. It is very difficult to see why, when the patient has been thoroughly oxygenated and when there is no muscular exertion how the modified technique could bring on a coronary attack. It is our belief that in the rare instances that this might occur, that the infarction should be considered a coincidental occurrence.

With the use of the Modified Technique, patients who previously could not be considered suitable for Standard Electro Shock Therapy can now be safely and effectively treated. On many occasions, we have treated patients with cardiac decompensation successfully after the internist has gotten the patient out of cardiac failure. On numerous occasions, we have treated patients with a history of coronary disease with infarction. In several instances, we have treated patients 10 weeks after an acute coronary infarction, when it was felt that the patient's mental condition was aggravating his cardiac status; namely, cases of agitated depression who were unable to relax or sleep. Recently we have successfully treated a seventy year old white married female, with auricular fibrillation. The patient, who was suffering from a severe agitated depression was treated while she was still fibrillating, and a complete remission of all her psychotic symptoms was obtained. These treatments were only instituted after consultation with a cardiologist.

With the Modified Technique, there is no convulsive seizure due to the use of Succinyl-Choline. The patients are flooded with oxygen prior to treatment, and following treatment, so that during the period of apnea, which occurs during treatment, there is a higher concentration of oxygen in the circulating blood stream than there was before treatment.

At this writing, we have given over 9,000 Modified Electro Therapy treatments at the Maine Medical Center with no skeletal injuries, cardiac complications, or any complications worthy of note. It is rather difficult after this series of treatments for me to understand why psychiatrists write papers about the dangers of Modified Electro Shock Therapy. We believe that the image of Electro Therapy such men have is that of the Standard Electro Shock procedure which they probably witnessed 10 or 15 years ago during their post-graduate training.

This author has no hesitation in making the flat statement that there are less complications when Modified Electro Therapy is used with a trained team including an experienced anesthesiologist than there are with the use of the anti-depressant drugs. The severe mental

confusion and organic psychotic reactions which were previously seen when Electro Shock Therapy was given, can easily be avoided by lessening the frequency of the treatment. In several persons in their eighth decade, we have found very little mental confusion when the frequency of treatments was reduced to three treatments in two weeks rather than the customary three treatments per week. Occasionally, a patient will complain of a headache or very rarely of being nauseated following treatment. This only occurs in apprehensive fearful individuals and in our opinion is not due to the treatment, but is due to increased nervous tension. Nausea is so rare that we do not see it more than once or twice a year. In many instances after the patient has been reassured, they no longer complain of headaches and/or nausea.

The advantages of Modified Electro Therapy in the treatment of the endogenous depression is that in over 95% of the patients the depression is aborted within two weeks. In fact, a large percentage begin to feel better and sleep better after the first treatment. With the use of the anti-depressant drugs, however, the literature states that there may be a latent period of from 10 days to 4 weeks or even a longer period of time before any amelioration of symptoms may be noted. We feel that this time factor, i.e., the rapidity with which the depression is aborted with Electro Therapy, is very important, first from an economic point of view, as we are usually able to get our patients back to work in 4 weeks, and also in so far as the mental anguish the patient is undergoing. Another important factor, is that during the period of treatment, which usually takes 2 weeks, and during the 2 weeks period of convalescence before the patient is discharged, he is not allowed to drive his car and is kept under very careful supervision by members of his family or friends to prevent the possibility of suicide. Recently, Martin<sup>7</sup> reported that "There are about the same number of successful suicides as there are deaths due to the communicable diseases in the State of Indiana. In 1957, it was reported that there were 16,760 suicidal deaths in the United States. This does not take into consideration, of course, many deaths reported as accidental, that actually were suicides. It is also felt that an appreciable number of our automobile deaths are produced by self-destructive reactions. The size of the suicide problem cannot be measured by successful attempts alone. The non-fatal attempts must be considered — The ratio of unsuccessful to successful attempts is not known: Various authors have placed this as high as 5:1. And if we consider the two we can see that we have a major problem — I am sure that if we had as many poliomyelitis cases and deaths as we have unsuccessful and successful suicides, we would experience a national panic."

A serious disadvantage with the use of the anti-depressant drugs is if the patient is not properly supervised during their administration and particularly if too much reliance is based on their efficacy, the danger of

suicide is ever present. This fact is particularly true in depressed patients who are agitated. As all psychiatrists know, this is the type of patient who is most potentially suicidal. Smith, Kline, and French in their literature, have stressed the point that if agitation is present, their anti-depressant drug (Parnate)<sup>®</sup> should be combined with Stelazine,<sup>®</sup> or Thorazine,<sup>®</sup> as they appreciate the fact that the anti-depressant drug does not have any effect upon the agitation.

It is a well-known fact to men experienced in the use of Electro Therapy that if patients respond favorably on one occasion to a course of Modified Electro Therapy, that subsequently, they have a 100% chance of recovery if they have a recurrence of their depressive symptoms regardless of the frequency of recurrence. There are many patients who will have one depression in a life time and never have another. Unfortunately, some people may have repeated depressions and may recur as frequently as every six to eight months. These patients can be very easily handled by the use of Maintenance Therapy, i.e., giving them one treatment when they are well every four to eight weeks prophylactically. There have been criticisms by both the medical profession and the lay public because one course of Modified Electro Therapy does not keep a patient permanently well. We have not heard the internist criticized because diabetics can only be controlled by the daily use of insulin and a dietary regime, or because the epileptic, can only be controlled by the daily use of an antiepileptic drug.

Wortis states<sup>8</sup> "The toxicity of Marsilid<sup>®</sup> makes it practically obsolete, but other monamine inhibitors may prove to be as equally dangerous. Ayd, who was quoted in the same article, thinks that not only Iproniazid (Marsilid), but Pheniprazine (Catron)<sup>®</sup> can be considered too dangerous to use, and adds that Isocarboxazid (Marplan)<sup>®</sup> and Phenelzine (Nardil)<sup>®</sup> can also have distressing, severe, and protracted side effects. He believes that the MAO inhibitors have been largely superseded by the anti-depressants, Amitriptyline (Elavil)<sup>®</sup> and Imipramine (Tofranil).<sup>®</sup> It should be noted that Imipramine (Tofranil) induces a marked drop in both systolic and diastolic blood pressure, of a range of 20 to 50 mm. Hg., in about half the patients with associated ECG changes, sometimes leading to cardiac complications. He also states that the MAO inhibitors seem best suited to the neurotic and atypical depressions.

Different medical drug houses have published literature stating that with the use of the anti-depressant drugs, all types of depressions are ameliorated, i.e., the reactive depression of Psychoneurotic origin the Schizophrenic, and the Sociopathic depressions as well as the endogenous or Psychotic group of depressions to a greater or lesser extent.

This has not been our experience. In a series of over 11,000 treatments, Electro Therapy has been found to be of specific value in the typical endogenous or

Manic Depressive Depression, and Involutional Melancholia. This fact was noted by this author in 1947.<sup>9</sup> The criteria for making the diagnosis of an endogenous depression that shows appreciable weight loss, self-accusatory, and self-condemnatory ideas, has difficulty eating and sleeping with suicidal tendencies and thoughts, and particularly those patients who have a family history of Manic Depressive Psychosis, respond within two or three weeks in this author's experience in over 95 per cent of the cases. As stated previously, if there is a neurotic overlay, the results obtained are not so specific.

Patients with reactive depressions of psychoneurotic origin do not respond favorably to Modified Electro Shock Therapy except for a period of two or three weeks immediately following the therapy when there may be some mental confusion due to treatment. Some of the severe reactive depressions which are treated with Electro Therapy become much more responsive when followed with psychotherapy. When using Modified Electro Therapy in Sociopathic and Schizophrenic depressions, one quickly realizes what limited value a course of Modified Electro Therapy may have. However, in the medical literature, particularly that supplied by all medical drug houses, anti-depressant drugs are supposed to be of definite value in any type of depression. However, we feel that one is intellectually justified in using Modified Electro Therapy in any patient who is actively suicidal. After many years of dealing with depressions, this author must admit that there are some cases of depression which are extremely difficult to classify prior to treatment. If one cannot treat an actively suicidal patient properly, one is at least justified in recommending institutionalization.

At a recent visit at the Augusta State Hospital where they deal primarily with the psychotic depressions, two of the senior physicians told me that they were ready to "Throw the psychic energizers and MAO inhibitors out of the window and concentrate on using Electro Shock Therapy in the treatment of depressions." The results at this institution with these drugs have apparently not been too favorable. From 15 year's personal experience with dealing with the different types of depressions, this author finds it difficult to believe that the same biological changes or causative factors can be present in all types of depressions.

The dangers and complications with the use of the anti-depressant drugs can be much greater than with the use of Modified Electro Therapy. Of course, the greatest danger is the potential danger of suicide which has been mentioned previously, and which is a serious danger if these depressed patients are ambulatory and not institutionalized or carefully supervised.

The inherent dangers from the side effects of the drugs themselves are quite serious. Recently the Upjohn Company has had to recall Monase<sup>®10</sup> a monamine oxidase inhibitor, because of the probable connection of the drug in patients developing agranulocytosis. The letter states that "The Upjohn Company in cooperation

with the Federal Food and Drug Administration is withdrawing Monase from the market." The Warner-Chilicott Laboratories recently wrote the following,<sup>11</sup> "The anti-depressant drugs — those that act specifically on depressions — have been in use for a little more than five years. Few have withstood the tests of effectiveness and safety."

Well-known complications of these drugs are symptoms of jaundice, liver impairment, and blood dyscrasias. There are numerous other side effects, such as hypotension, restlessness, insomnia, dizziness, skin rashes, blurred vision, and, symptoms too numerous to mention, which can be noted by reading any of the literature provided by the anti-depressant drug houses. Exclusive reliance on drug therapy to prevent suicide is unwarranted unless the patient is institutionalized. Some of the contraindications from the use of the anti-depressant drugs are impaired liver and renal function and recent myo-cardio infarction, and coronary disease. However, these patients do not have to be excluded when Modified Electro Therapy is considered.

A colleague recently reported a case of hepatomegaly associated with Marplan,<sup>®</sup> the successor to Marsilid, which had to be removed from the market because of toxicity.<sup>12</sup> Hepatomegaly, hypotension, and other associated findings receded within five days after the withdrawal of the drug, Marplan. How much permanent damage was done to the liver is problematical. This patient promptly recovered with a course of Modified Electro Therapy.

L. Gahagan<sup>13</sup> writes in a letter to the editor of the American Journal of Psychiatry, criticizing "The partial replacement of a highly effective and safe mode of treatment (ECT) with an inferior mode of treatment (anti-depressant drugs) one wonders if there are other such instances of therapeutic regression — it is hard—at least for me—to imagine anything more deplorable than the failure to use early and adequate ECT in involutional depression — even the most optimistic 'claims' for anti-depressant drugs are short of the 'attainments' of Electro Shock Therapy in the treatment of the involutional and other severe depressions." It is our feeling that in the true endogenous depression, when the results are so clear-cut and the depression can be aborted in such a short period of time that it certainly does constitute a therapeutic regression to replace a tested therapeutic remedy by an experimental drug.

Another very pertinent fact which should be brought to the attention of the medical profession in the State of Maine is that the suicide mortality rate for the United States of America as a whole is 10.6/100,000 population. In the State of Maine it is 12/100,000, one of the highest suicide rates in the Nation. This fact is due partially, no doubt, to the shortage of psychiatrists in this state, and also due to the reluctance of many individuals to avail themselves of psychiatric help when it has been advised. When these startling figures are brought to the attention of the medical profession, it

would behoove us all, to make an effort to try to reduce this needless loss of life.

#### SUMMARY

1. Unmodified Electro Shock Therapy is an out-moded procedure. Its use exposes a patient to unwarranted and unnecessary hazards which are readily and safely prevented by the use of the Modified Technique.

2. Modified Electro Therapy is still by far the safest, quickest, and most reliable form of treatment in the severe depressions—particularly those of the psychotic type, and in those cases where the patients show agitation.

3. The inherent complications with the use of anti-depressant drugs are potentially dangerous. They may be lethal, and are much more serious than any complications experienced with the use of Modified Electro Therapy.

4. The physical contraindications with the use of anti-depressant drugs i.e., liver, renal, or cardiac impairment are not contraindications to the use of Modified Electro Therapy.

5. Definitive steps should be taken by the Medical Profession in Maine to prevent it being classified as a state with one of the highest suicide rates in the Nation. This can readily be done by the more frequent referral of depressed patients to proper clinics, mental institutions, or psychiatrists.

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# Casework Services To A.D.C. Families\*

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In this session on "Services to Families and Children," which your Program Committee has planned, the specific focus assigned to me is that of "Casework Services to A.D.C. Families."

The same words, spoken or written at different times, have different meanings and connotations. I am sure that members of the committee had in mind different ideas than they might have had, had they been making these arrangements for a session with the same title twenty-five years ago at the point when Congress had just passed the Social Security Act, a part of which authorized the Aid to Dependent Children program.

A quarter of a century is a generation of time. Boys and girls, five years of age or more, whose mothers received assistance in those early years of the program, are now men and women in their thirties. Most of them are probably parents now themselves. You know some of these young men and women personally, and so do I. I number some of them among my friends. I have also seen young people who applied for admission to schools of social work, and some who were admitted, whose mothers had received A.D.C. Some of these young people wanted to enter the profession because they had learned from their own experience how helpful social work can be. Others approached schools of social work with a highly negative commitment. They were determined that they wanted to be social workers so that *their* clients, at least, would *not* be treated as they felt their families had been treated.

It is also quite likely — and it would be statistically improbable for this not to be true — that a few of those early A.D.C. children are now parents who are receiving public assistance themselves. However, it seems altogether safe to say that the vast majority of these former A.D.C. children are now self-supporting adults. They are working at their jobs as plumbers, electricians, farmers, nurses, salesmen and saleswomen, stenographers, teachers, laborers, business entrepreneurs, janitors, lawyers, grocery clerks, doctors, sewing machine operators, assembly line workers and bookkeepers. They are paying their taxes toward the continuation of the program as we now know it. Quite probably, the vast majority of these citizens are quite undistinguishable from the rest of the great anonymous body of tax payers.

A quarter of a century is a long time, however, and in that period a great many changes in programs have come about. Any social welfare program is influenced by a variety of forces which affect the changing shape

and form it takes. This would be true whether the program is public or private, although in identifying these factors, emphasis is placed on factors affecting a public program such as is our concern here. In the first place, a program comes into being because some social need existed. The program, as established, is a reflection of (1) the legislators' interpretation of the social need, (2) their idea as to how the need can best be met, given the resources available, and (3) their opinion of the persons having those needs. Secondly, the program is influenced by the varieties of problems brought by the persons for whose benefit the program was established, whether or not these needs and problems had been anticipated. Thirdly, the program is influenced by what the various administrators *think* was the intent of the legislators, and fourthly, by what they *hoped* was their intent, and fifthly — sometimes — by what they *wished* the intentions of the legislators had been, even though they knew perfectly well that the legislators had no such intention in mind! Sixthly, the shape of the program is influenced by the quality of skills, understanding and philosophy of those persons within the organizational hierarchy below the top administrative echelons. Finally, the program is influenced mightily by public opinion about its aims and accomplishments, strengths and weaknesses. Public opinion, vocally expressed, goes back to the ears of the legislators and brings about praise or denunciation of the program, increased or decreased appropriations, demands for investigation or appraisal, and sometimes changes in the law. Then the whole cycle of the effects of forces and counterforces is ready for repetition. In the meantime, of course, and most important of all, changes in the general social climate have come about, and new social problems have arisen which influence the shape and form of the program.

In all of the above, no mention has been made of the further complication that in a program such as ADC, it is not one legislative body, one administrator, and one set of personnel, one source of public opinion, but rather with local, state and federal involvement, each of these kinds of forces may operate at each level — and sometimes in contradictory direction at the same time.

It is not necessary with this group to belabor the point that changes in the social scene, extrinsic to the ADC program itself have brought about tremendous changes in the kinds of problems characteristic of those families receiving this form of public assistance. Increased coverage of Old Age and Survivors' Insurance both in terms of numbers of persons receiving benefits and in terms of increased amounts of benefits has de-

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creased the need for ADC in behalf of children in families where the breadwinner has died. Both the number and the rate of illegitimate babies in the population have increased sharply. Whereas in 1938 only seven out of every 1,000 unmarried women between the ages of 15-44 had an illegitimate child — that is about one in every 143 — in 1957, 21 out of every 1,000 — or about one in every 49 — had an illegitimate child. An estimated 200,000 illegitimate babies were born in the United States in 1957.<sup>1</sup> An estimated 393,000 women secured divorces in 1959,<sup>2</sup> and about half of the divorces granted involved children.<sup>3</sup>

The ADC program reflects these social problems. An increasing proportion of the children being served by the program are those whose fathers are absent from the home because of separation, divorce or the unmarried status of the parents. To state this, however, is not the same as falling into the logical error of mistaking effect for cause, and saying that the existence of the program caused these social problems. Furthermore, the numbers of children whose parents are unmarried, separated or divorced, who are being supported by the program, is vastly less than the numbers of such children whose mothers are not receiving public assistance in their behalf.

Nevertheless, the effect of these social phenomena upon the ADC program has been succinctly summarized by Alvin Schorr. He writes, "What has happened as a social accident, is that ADC — designed in one social era and moving into another — now operates selectively to serve people of whom social problem and community disapproval are characteristic."<sup>4</sup>

Quite probably, all of you here have seen and heard evidence of such disapproval directed against the persons served, or against the agency providing the service or against yourselves as representatives of these agencies. These are some of the kinds of complaints you hear. "She wastes the money on liquor and foolishness. The kids don't get the benefit of it," or "she ought to be forced to take care of her kids" or, contrariwise, "Her kids ought to be taken away from her." or "He says he's too sick to work but I notice that they keep on having a baby every year," or "Why should I pay taxes to support that tramp and her babies. She just keeps on having babies so she can stay on relief," or, "If these men can't look after their families, they all ought to be put in jail for a while," or, "If relief wasn't so easy to get, people wouldn't be so lazy."

It is within this context of public criticism and current program problems that "Services to Families and Children" and "Casework Services to ADC Families" are being considered. In this context "services" has come to mean social rehabilitation in behalf of persons with a wide range of adjustment problems. Furthermore, there is the connotation that "services" refers to something *other than* financial assistance.

Only recently has the public welfare field itself faced up to the implications of the fact that a considerable

number of the members of these partial and broken families are persons who, in addition to economic need, do have serious problems of social adaptation in their roles as spouses, parents, employees, and members of the community. It is important that public welfare personnel at all levels of organization within each organization and at all political levels — local, state and Federal — acknowledge that the program has changed and that, to some extent, the program has become one through which the community pays a part of the economic and social cost arising out of the problems of maladaptation of some of its members.

Such acknowledgement rewards the administrator and his staff by relieving them of the need to use their energies defensively and instead frees their energies for the purpose of finding ways and means to provide better service. However, such acknowledgement also carries a penalty if there has been any tendency on the part of the administrator to assert that the business of his department was only to provide financial assistance — not to provide help with problems of social maladjustment. Further, it makes thoroughly untenable the position that if problems of social maladjustments do come to the workers' attention, they are responsible only for referral to the appropriate private agency. In the first place, in many communities, there are no private agencies, appropriate or otherwise. In the second place, even in cities where there are private agencies, it is doubtful if these agencies could meet the need. In New York City, for example, if all the private family agencies devoted all their time and energies to none other than public assistance recipients, the available services would fall far short of even the most modest estimate of the extent of the need.

There is a third reason why such a policy of referral is unfeasible, based on the type of problems and personalities of the persons needing help. I trust, of course, that none of you will interpret the following statement as meaning that it is applicable to all persons receiving ADC. Obviously, a diagnostic evaluation must be based on the specific individual in his own particular situation. Nevertheless, it is not inappropriate to suggest that many of the persons whose problems of maladaptation are most grave are least likely to accept referral. By and large, these are not the agonizing neurotics who have some conscious awareness of tensions, maladjustment, strained relationships, anxieties; instead, these are often the individuals who are very anxious indeed, but so deeply uncertain of themselves that they cannot even bear to become aware of these feelings and who act out their impulses to find momentary satisfactions and to rid themselves of tensions. Furthermore, they are often the individuals who can see value in a casework relationship only if the worker has the capacity to give them tangible help, either in direct provision of material needs or in helping them learn how to reap rewards for their own behavior by helping them acquire skills in dealing with the things

and the people in the environment in which they live, thus giving them more useful and appropriate ways of protecting themselves against their sense of helplessness and worthlessness and their own self-destructive tendencies. The public assistance worker best fits the description of the kind of individual whom persons with these kinds of problems can regard most readily as potentially helpful persons. The policy of referring clients elsewhere for service, when there is no available service elsewhere or when the client's own problems are such that he cannot accept referral, is in reality a buck-passing "no-service policy," not a service policy at all.

The public welfare administrator's plea that the personnel at his disposal are insufficient in number and in training to carry out these new kinds of responsibilities becomes quite unconvincing if it be weighed against the fact that public assistance agencies have more personnel available than any other type of social welfare agency and that the administrators' "too few" must be balanced against the "far, far fewer" available elsewhere. Although this paper concentrates on the idea of problems in families, I wish to make it clear that I do not believe that all families receiving public assistance have serious problems of maladjustment. But I would take it that these families who get along fine and whose only need is economic are not really the families about which this group would be troubled.

At the risk of being regarded as reactionary, I should like to suggest that it would be regrettable indeed if public welfare agencies were to embrace the so-called "other than income maintenance services" in order to escape the knotty problems which yet remain with us in meeting economic need in a constructive manner. This may be put even more bluntly — the time has not yet come for administrators to relax and say it is now up to workers to give individualized services. Indeed, for the very reason that public welfare workers represent such a wide range of background, education, experience and skill, it is all the more important that the policies which pertain to financial assistance be constructive in themselves. Good policies can insure at least a minimum quality of service, but it is difficult indeed for an individual worker to render good service, however skilled he may be if the policies within which he operates are themselves destructive of the family's self respect. Therefore, the deepening awareness of the extent and gravity of the social adjustment problems of ADC families should bring more, rather than less, attention to administrative policies.

On this basis, it is perhaps well then to mention some of those unsolved questions of law and of administrative policy. The Advisory Council on Public Assistance made the following statement and recommendation:

"Under the existing provisions for aid to dependent children, Federal grants in-aid are available to the states only for the assistance of children deprived of support or care because of the absence, death or incapacity of one

parent. As an ironical result in many states, destitute children living with two able-bodied parents are actually penalized. On the premise that a hungry and ill-clothed child is as hungry and ill-clothed if he lives in an unbroken home as if he were orphaned or illegitimate, the program for aid to dependent children should be expanded to include any financially needy children living with any relative or relatives "in a place of residence maintained by one or more of such relatives as his or their own home."<sup>5</sup>

Were this recommendation enacted into law, it would radically decrease the proportion of ADC children living in partial or broken families, but this should not delude the administrators into believing that these problems, thus somewhat hidden, were solved. Also there would be some added casework dimensions in providing financial assistance through ADC since the number and proportion of households in which there was a potential breadwinner would be increased, but of course some of these families are now receiving general assistance.

Ellen J. Perkins has called attention to the simple basic matter of the insufficiency of the grant. Not only are some states not meeting the full amount of budgetary needs as computed by their budgets, but also the budgets in use are usually inadequate. Using a formula based on the United States Department of Agriculture food plan to compute a budget, Dr. Perkins estimates that in the states in the Northeast region, ADC budgets as currently computed meet only 79.5% of the need of client families.<sup>6</sup>

Referring again to Schorr, he points out that whether reckoned by percentage of increase in amounts of grants over a period of years or in average amount of grant per recipient, the ADC family fares less well financially than recipients in other Federally aided programs. In the 25 states whose laws include maximum grant provisions in all categories, this maximum "turns out to be as high for one person in the other three Federally aided categories as they are for an adult and one child in ADC."<sup>7</sup> This phenomenon, he believes, is a reflection of the community disapproval of ADC recipients, referred to earlier.

The manner in which the grant is made may serve either to weaken or strengthen family solidarity. We have only begun to dare to look at the possibility that maybe, as some of our critics have said, some practices do encourage the idea of the mother-children concept of family as when, for example, the mother is the payee of the ADC check even when the father is in the home. Sometimes the incapacitated father gets a separate A.D. check. If this father, even before incapacitation, had found it difficult to meet the economic and emotional responsibilities of parenthood, then it is a short step between this method of handling grants and his coming to regard himself as a kind of over-aged adolescent who is being granted a spending allowance rather than being helped to see himself as the continuing head of the family, even though incapacitated.

In addition to policy, the worker's attitude is another

force affecting the sense of family unity. The ADC worker may have become so accustomed to dealing with partial and broken families in which the mother is the head of the household that when the worker does encounter a complete family, he is baffled about what to do about the father and gives the impression that the whole matter could move along in a more comfortable routine if that extraneous man were not there.

A third matter pertains to policies and practices when there is an income derived from employment. I should like to put the problem in the broader context of attitudes about dependency, independence and work. Some of the social work literature dealing with the questions of what it means to a client to apply for assistance and to receive assistance is contained in the classic articles and monographs which were written within the depression era. I think it is safe to say that it makes little difference whether those pieces of literature were the Epistles written to the Smith College School of Social Work students or the Epistles to the Chicagoans or the Pennsylvanians or the New Yorkers. In all of them, and they continue to be widely read — there is the underlying theme that it is an extremely difficult experience for an individual to apply for public assistance and to be a recipient of assistance. At the time these articles were written, this was undoubtedly true in the overwhelming majority of cases. For that was the era in which the depression, like some great monster, grabbed millions of citizens by the neck, hoisted them in the air and held them dangling — but kicking and struggling to get back to the solid footing of job, wages, self-support, independence.

Most people do want to be independent and self-supporting individuals. Their previous life experience has been reasonably satisfying, has brought successful achievements, and has prepared them for finding satisfaction in independence. However, these classic pieces of literature either fail to make explicit, or we fail to pay proper heed to the muted point that sometimes individuals have *not* had these developmental experiences and that some persons reach chronological maturity without a matching capacity for mature self-direction and acceptance of responsibility.

By this round-about route, I come back to the question: What are the effects of our policies concerning income derived from employment? Methods for computing budgetary need for these families in which there is a working member often operate in such a way that the family is little better off financially than the family totally dependent upon financial assistance. If the father or mother secures employment which nets an income equal to or only slightly more than the needs as computed by public assistance standard, that family is expected to get along without further assistance. There are sometimes slight increases in the computation of the budget because of extra expenses allowed for a working person but these are often very small indeed. Such policies can only be based on the psychological

premise that all individuals prefer working to not working, just for the sheer joy of it. I doubt whether this is the case, our classics in social work literature notwithstanding. This promise fails to take into account that many of the persons now receiving assistance have had little training or skill for work which is rewarding in and of itself. They have had few successes in their efforts to achieve. Their work histories are spotty. If these persons are to be helped to become self-supporting, they need to experience tangible rewards for working. The implication of this is that when an adult in the family secures employment, a more generous budget should be used to determine whether the family continues to be eligible for supplementary help so that working and earning money carries with it the advantage of the family being able to enjoy a better standard of living.

There is another unsound feature of a stringent policy. Erik Erikson, writing about the stages of development in children points out very charmingly that when a little boy begins to walk, he has become a different person, and enjoys a different status. He can now regard himself as "He who walks by himself." So, too, the individual who takes a job must be able to think of himself as "He (or she) who has a job." And he or she who has a job is expected to be different. Fellow workers expect that he will contribute to the kitty for a fellow worker's party, stop in for a beer on the way home from work, indulge an occasional impulse which is unscheduled and unbudgeted. More important, the employed parent hopes to be able to respond to the children's requests for treats in a more generous manner than he was able to do when the money came from the public welfare department. When a father's earnings are only marginal and where previous experience and personality characteristics make immediate gratifications more important than long range goals, such advantages from working are particularly important. If they do not accrue, it is not at all surprising that some of these fathers are counted as "separated" in the ADC caseload. They derive more gratification — and self-esteem — from "visiting" their families — unbeknownst to the P.A. worker of course. They arrive at their homes, laden with gifts for their children and the potential for providing sexual pleasure for their wives. Then, unloaded, they leave. This is more satisfying than remaining at home for no matter how hard they work they still find themselves in the position of being penurious fathers. This might be particularly true if, remaining at home, wives remind them that they can earn "no more than what the relief used to give us."

The same principle of instituting policies in which it "pays" a member of the family to work would hold whether the employed person is a father or a mother, and particularly if it be an adolescent boy or girl who is expected to contribute to the family's budget when employed. Good attitudes toward work are not likely

to be established in the young person just beginning his employment career if he feels that he is working only to save money for the welfare department.

While all of these approaches are dependent upon administrative policies, and back of this, public support, only the individual worker can implement them effectively as he deals directly with the families whom he serves.

Other services to families are more clearly dependent upon the initiative, sensitivity and skill of the individual worker.

A worker cannot sensibly undertake helping a client achieve more effective social functioning and greater personal satisfaction without taking into account the client's bodily capacities and the quality of his health. All forms of social functioning involve some aspect of bodily functioning. Since the body is a unitary whole and is not made up of detachable parts and appliances, it is necessary to regard the total quality of bodily capacities and of health as being involved, either directly or indirectly, in all aspects of functioning. We expect ADC mothers to mother their children — and while mothering may have its spiritual aspects, it also has in it a lot of earthy bending, stooping, cooking, cleaning, washing of dirty faces, picking-up-after, being patient, hugging, kissing, occasionally even spanking — and all of these things are a lot harder to do with aches and pains. Ordinarily, bodily ailments direct the individual's attention and love back into himself and dam up the outward flow of love and energy.

One of the shocking findings of Future Citizens All, the research study written some years ago, was that children in ADC families had been provided with fewer health services than children in families of comparable economic status not receiving ADC.<sup>8</sup> Somehow or other, the workers involved with these families had lost their opportunities to help families make use of health facilities. One can surmise that some of these children are reaching adulthood with a physical defect or health problem which might have been corrected in childhood but which, carried into adulthood remains a handicap to effective functioning.

It is to be hoped that present day social workers are not losing these opportunities to be helpful. However, simple referral to clinics and other medical resources, or assurance that medical costs can be included in the budget may not be enough. Some clients need to be taught how to use clinic facilities in behalf of themselves or their children. Some are too frightened of the complex organizational structure of a modern hospital to risk getting into its clutches. They need advance preparation and explanation of the things which will happen to them which, if not understood, are both unnerving and mystifying. Dr. Beatrice Bishop Berle discussing the use of health facilities by Puerto Ricans in New York City points out the immense difference in the way in which the same episode or series of episodes are perceived by the hospitals'

personnel and by the patient himself. Sometimes patients are referred to the hospital clinic system by welfare centers to determine whether there is any organic problem which interferes with employability. At the hospital, the patient goes through one clinic after another. He has appointments on different days, at different hours in different parts of the huge hospital buildings and is seen in each clinic by different sets of medical personnel, each concentrating on different parts of his anatomy. For purposes of laboratory examination, he is required to produce upon demand sputum, blood and urine. Finally, after all parts of him have been poked, probed, examined, listened to and looked at, and after a series of strange doctors have talked to each other about him across his prone form in words he does not understand, he is discharged from the hospital with a clean bill of health — all findings are negative. But so far as the patient is concerned, he has no idea why he was sent from one clinic to another. He is sure that the seriousness of his ailments is directly proportional to the number of doctors who saw him. He emerges from the hospital thoroughly convinced that he is a very sick man indeed.<sup>8a</sup>

Sometimes the client needs the worker's help, or the help of the medical social worker in the clinic or hospital as an intermediary to understand what the doctor really meant by the medical phraseology.

To a certain extent, all of us are victims of this era's folk medicine as practiced by television commercials and the drug and vitamin advertisements. Polysyllabic words are today's magic medicine. We mouth them without any real understanding at all of just why our vitamins and cold remedies are so much better than Brand X.

Clients are sensitive to our reasons for being concerned with their health, discriminating between a genuine concern for their well being and an interest based only on the question of their employability. Naturally, acquainting the client with medical resources, helping him make use of them and helping him understand the meaning of medical findings are not always going to be effective. There may be a neurotic investment in physical ailments and complaints which counteract these efforts but until the worker has tried these practical measures, there is no basis for reaching that conclusion.

Other types of service might be classified under the general heading of improving the clients' capacity to deal with reality problems by expanding his knowledge and improving his skills.

A considerable number of families receiving public assistance might be classified as "hard-to-reach" families, "multi-problem" families, "resistive" families. In recent literature which describes methods and techniques for helping such families, two of the themes are these: (1) Where the worker takes the time and trouble really to become acquainted with these clients and the way in which they manage their ordinary daily tasks, it be-

comes evident that they are woefully lacking in the kinds of knowledge and skills which are usually taken for granted; and (2) many of these so-called "hard-to-reach" persons are able to use help with problems of relationship only *after* the worker has proved himself to be a valuable ally in helping them with these problems of everyday living.<sup>9,10,11</sup>

Two of the most important intellectual tools developed by man are writing and arithmetic. Some degree of competence in both are almost essential for getting along in a modern community, yet large numbers of persons in our midst have little or no knowledge of spoken English and there are many native born Americans whose reading knowledge of English is painfully rudimentary. Reading an employment application, a recipe, the directions for the use of household equipment are difficult, if not impossible tasks. Perhaps an even greater number of persons are "illiterate" in arithmetic — including some college graduates. Not long ago, I asked a graduate student what interest rate she was paying on a loan and she did not know.

Probably you all know clients who have gotten into acrimonious tangles involving arithmetic. For example, a client with perfect sincerity, accuses the landlord of trying to cheat him. The client has been paying two weeks rent each of the twelve times she received her semi-monthly assistance check so now the landlord tells her she is two weeks in arrears — which she is, but this is incomprehensible to her if she does not know that a year has in it 24 half month periods but 26 two-week periods of time.

Or a client is astonished at how much an article costs when the worker helps multiply the "small weekly payments" by the number of weeks she is expected to pay.

Last semester, I read a case record which demonstrated how a social work student helped a client with her budget problems. There were discussions about food shopping, and the student figured out with her on the basis of comparative prices how she could save by shopping differently. It was a pleasure to read the case entry where a few weeks later the client with great satisfaction told her worker that she had saved \$4.00 by shopping at the supermarket. More important, she very proudly said that she had been talking with her neighbor about this too, and that this week she was going to show her neighbor how it was done. The woman was not just using the grant more efficiently, she had become a slightly different person. Inside of her as a part of herself she now possessed knowledge and skill not there, or not used, before. Paraphrasing Erikson again, she was now "She who is wise in the use of money."

Some of the literature referred to above speaks of the great meaning which can be carried by giving help to women clients in simple suggestions about cooking. Again, the casework aim is not to give a cooking lesson — it is to help that wife and mother be "She who knows

how to please her man with tasty food," "She who knows how to take good care of her children."

The same student, cited above, used similar techniques to help a man who was fearful of applying for a job because he didn't know what prospective employers would say to him about his having been in jail. Together, the worker and client thought about the kinds of questions the employer would ask and what answers would be suitable. The student even asked what kind of clothes the man usually wore when he went out looking for work. Like so many persons needing assistance, he had had little experience with success. Weak with words, he was inclined to communicate with his fists. Uncertain of his own capacities as a human being, he was prone to respond to difficult situations by proving he had great brute strength. He needed to be "rehearsed" for the successful use of other methods. P.S. He got the job.

Many of our clients, in their encounters with landlords, credit managers, teachers, doctors, nurses- and, sadly sometimes with social workers find themselves baffled, confused, beaten-down and overwhelmed by the force of the superior word-power of these persons in positions of authority. They come away from these encounters feeling they might better have "stood in bed."

Those clients who have come into the community from different kinds of settings have even greater problems for they are even less likely to possess needed knowledge and skills. The person moving from a rural to an urban economy, or into a setting where a different language is spoken is a "displaced" person, for all practical purposes, and regardless of his nationality or citizenship status, he may need as much help as though he were living in a foreign land.

Unless the worker is laboring under the delusion that casework consists of nothing but sitting here with the client and saying "Um-m-m" at regular intervals, he will realize that it is incumbent upon him to learn the art of speaking directly, simply and understandably to clients. Sometimes this may involve the personal wrench and sacrifice of putting aside false and foolish pride in knowing technical words and in using abbreviations which are part of agency jargon but which only confuse and bewilder clients.

The important point in all of the illustrations given above is that the lack of a specific piece of knowledge or skill, or the possession thereof, is not a thing apart from the rest of the personality. These limitations or attributes are a part of the total person and affect not only the performance of the task for which the knowledge and skill is needed but also affect the individual's self esteem and his relationship with others.

Another type of service is implicit in some of the above statements — that of helping the client become reintegrated into the framework of community activities and institutions — specifically non-social work associations. Isolation from the community sometimes

characterizes the "problem family." Therefore, although many of the clients might have comparable problems which could be dealt with on a group basis as, for example, language classes, homecraft groups, and so forth, it is *not* suggested that such groups be set up exclusively for public assistance recipients. Rather, they should be encouraged to become a part of other groups. The aim is to reintegrate into community activities — not to establish a public assistance fraternity or sorority.

A criticism sometimes leveled at the ADC mother is that she associates with the wrong people. In regard to this, the searching question which we must ask ourselves, and which the community ought to ask itself is, whether she has been offered any other associates who would accept her as whole-heartedly, provide her with as much gratification and be as ready to help her in time of emergency as those individuals whom we glibly and quickly label as "undesirables."

Clients need to know from us that we do not think that receipt of public assistance deprives them of their right to find enjoyment in life.

None of us is so naive as to believe that the services suggested above are going to solve all of the problems of low esteem, impulsive acting-out behavior, distorted concepts of reality and of self, confused sexual identifications, and problems of relating to others which are sometimes manifested by marital conflict and unmarried motherhood. The suggested casework services, to recapitulate, are these, (1) providing a grant adequate in amount to provide a standard of living compatible with standards of health and decency; (2) providing it in such a way that it will have a constructive rather than destructive effect upon family unity and stability; (3) making medical services available and by educative techniques within the casework relationship, making it possible for the client to use these facilities; (4) increasing the clients' capacity to deal with the every day problems of living by helping him expand his knowledge and skills; and (5) reintegrating the client and his family into the framework of community activities, including fun and recreational facilities.

While, as admitted, these measures will not remedy all problems of relationship, it is contended that some of such difficulties spring from the problems created by these inadequacies which the above suggestions are intended to correct. A completely obvious truth, often overlooked, is that when a person quite realistically is unable to function effectively as a wife, homemaker and mother, or a husband, father and provider, because of lack of funds or equipment, handicapping health conditions or defective knowledge or skills, then the best way to help that person *feel* more adequate is to help him *be* more adequate. His improved capacities and better feeling about himself cannot help but influence his relationships.

It is recognized that the above services require time, skill, and often much patience, for progress may be slow. The worker's task is to ask himself in each situation "Are any of these handicapping conditions present in the person or in his environment and, if so, what can I do about them?" This means that he must be acutely aware of cues to their presence, to be extraordinarily alert in listening to the client so that between the things the client says he may "hear" the partly hidden acknowledgements of need, the gaps of knowledge, the absence of skills, the confusions which exist. Further, these services require from the worker a respect for the client's right to find pleasure and satisfaction in living as well as an awareness of the client's responsibilities. They require that the worker have a true interest in helping which the client is sure to sense, and that he has a nice sense of timing so that the spoken suggestion, the reaction to the client's expression of feeling, the piece of information which will correct a misconception, the offer to show the client how, all will be given at the point of the client's readiness to receive. The worker needs a peculiar combination of being vividly imaginative in a very practical way so as to be able to understand and sometimes anticipate the nature of the client's feelings, problems and the specific difficulties he has in performing tasks and, at the same time, be able to think of very practical concrete ways to be helpful. All of these half dozen or so attributes of the worker require the best we have to give of professional knowledge, skills and attitudes and of ourselves. Hopefully, they enable the clients to think of us quite simply as "a friend in need" as well as their workers.

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# From the Secretary's Notebook



## 109th Annual Session House Of Delegates

*Continued from August Issue — page 205*

### MAINE MEDICAL EDUCATION FOUNDATION

VOTED that members of the Maine Medical Association contribute (voluntarily) \$25.00 to the M.M.E.F. for this year, 1962.

### PROPOSAL FROM COMMITTEE ON RECRUITMENT, AID AND PLACEMENT

VOTED that the House of Delegates accept in principle the proposal (relative to establishment of a Loan Guarantee Fund) as outlined by Doctor Charles W. Capron of Portland, further details to be worked out by the Committee on Recruitment, Aid and Placement of the Maine Medical Association and a Committee of the Maine Banker's Association with their respective legal counsel.

### RESOLUTION REGARDING "IV THERAPY"

This resolution was presented by Doctor Thomas A. Martin of Portland, for the Liaison Committee of the Maine Medical Association and Maine Nurses' Association. The resolution as accepted follows:

WHEREAS, the administration of drugs, fluids, plasma expanders and blood by the intravenous route have become a common procedure, and

WHEREAS, nurses have traditionally administered medications to patients on the order of physicians, and

WHEREAS, it is common practice in Maine for some registered professional nurses who are duly qualified in vena puncture techniques and educated in potential hazards, to administer intravenously substances ordered by physicians,

NOW, THEREFORE, BE IT RESOLVED: That the Maine Medical Association affirms that intravenous administration of substances ordered by physicians constitutes an accepted and recommended duty under the practice of nursing when done by qualified registered nurses of proven competency in hospital or affiliated institutions.

### PROPOSED LEGISLATIVE BILL SUBMITTED BY COMMITTEE ON CLINICAL HYPNOSIS

This proposed legislation as approved by the House is:

#### AN ACT TO PROTECT THE PUBLIC FROM THE USE OF HYPNOSIS BY UNQUALIFIED PERSONS

Sec. 1: If any person shall hypnotize or attempt to hypnotize any person, he shall be guilty of a misdemeanor. But this section shall not apply to hypnotism performed by legally accredited doctors of medicine, osteopathy, dentistry, and psychology; provided, however, that such qualification is in accord with proper medical, dental and psychological professional requirements, and is not used for purposes of

entertainment. Psychologists shall utilize hypnosis for therapeutic purpose only in accordance with existing laws.

Sec. 2: Misdemeanor, for which no punishment or no maximum punishment is prescribed by statute, shall be punished by fine not exceeding five hundred dollars or confinement in jail not exceeding twelve months, or both, in the discretion of the jury or judge.

Sec. 3: The Maine Board of Registration of Medicine for licensure and regulation of medical doctors, and the similar licensing bodies for osteopathic physicians, dentists, and accredited psychologists, shall administer and enforce this Act and may designate the Office of the Attorney General, which may appoint any legally qualified medical practitioner for the purpose of making any investigation or inquiry necessary therefore.

Sec. 4: Every prosecution under this Act shall be commenced within one year from the date of alleged offense.

Sec. 5: The restrictions of Section 1 do not apply to any bona fide student registered in a course leading to qualification in one of the professions named in the Section practicing hypnosis for the purpose of study under the instruction and supervision of a legally accredited doctor of medicine, dentist, or psychologist.

Nothing in this Act shall prevent the exhibition, demonstration of performance of hypnosis for scientific or research purposes, or for the purposes of instruction by and for any medical doctor, osteopathic doctor, dentist or doctor of psychology or any persons who are members of the nursing profession, or in courses leading to qualifications in a nursing profession.

### RESOLUTION SUBMITTED BY DOCTOR PAUL H. PFEIFFER

WHEREAS, the average working registered nurse earns \$1,000.00 less than the average woman working in industry, and

WHEREAS, the nursing profession represents an essential and dedicated part of the medical team, and

WHEREAS, an acute and desperate nursing shortage is developing, causing entire hospital wards to remain vacant for lack of nursing personnel,

BE IT, THEREFORE, RESOLVED by the Maine Medical Association that (1) the Maine Medical Association inform the Nursing Association that it is aware of the nurses' economic plight and (2) the Maine Medical Association will do what it can to improve this unfortunate situation.

VOTED to accept the resolution as read.

*Continued on Page 232*

*the first comprehensive  
regulator of  
female cyclic function*

# ENOVID®

(brand of norethynodrel with ethynylestradiol 3-methyl ether)

## THE BASIC ACTION

ENOVID closely mimics the balanced progestational-estrogenic action of the functioning corpus luteum. This action is readily understood by a simple comparison. In effect, ENOVID *induces a physiologic state which simulates early pregnancy—except that there is no placenta or fetus.* Thus, as in pregnancy, the production or release of pituitary gonadotropin is inhibited and ovulation suspended; a pseudodecidual endometrium ("pseudo" because neither placenta nor fetus is present) is induced and maintained.

Further, during ENOVID therapy, certain symptoms typical of normal pregnancy may be noted in some patients, such as nausea—which is usually mild and disappears spontaneously within a few days—breast engorgement, some degree of fluid retention, and often a marked sense of well-being. There is no androgenicity. ENOVID *is as safe as the normal state of pregnancy.*

## THE BASIC APPLICATIONS

**1. Correction of menstrual dysfunction.** *Emergency* treatment of severe dysfunctional uterine bleeding is promptly effective following the administration of ENOVID in larger doses. *Cyclic* therapy with ENOVID controls less severe dysfunctional uterine bleeding. In amenorrhea *cyclic* therapy with ENOVID establishes a pseudodecidual endometrium providing the patient has endometrial tissue capable of response.

**2. Ovulation suppression (to suspend fertility).** For this purpose ENOVID is administered *cyclically*, beginning on day 5 through day 24 (20 daily doses). The ovary remains in a state of physiologic rest and there is no impairment of subsequent fertility. When ENOVID is prescribed for this *cyclic* use over prolonged periods, a total of twenty-four months should not be exceeded until continuing studies indicate that its present lack of undesired actions continues for even longer intervals. Such studies are now in their seventh year and will regularly be reviewed for extension of the present recommendation.



*...unfettered*

**3. Adjustment of the menses** for reasons of health (impending hospitalization for surgery, during treatment of Bartholin's gland cysts, acute urethritis, rectal abscess, trichomonal or monilial vaginitis), or other special circumstances considered valid in the opinion of the physician. For this purpose ENOVID may be started at any time in the cycle up to one week before expected menstruation. Upon discontinuation, normal cyclic bleeding occurs in three to five days.

**4. Endometriosis.** *Continuous* therapy with ENOVID corrects endometriosis by producing a pseudodecidual reaction with subsequent absorption of aberrant endometrial tissue.

**5. Threatened and habitual abortion.** ENOVID should be used as *emergency* treatment in *threatened abortion* although symptoms may occur too late to be reversible. *Continuous* therapy with ENOVID in *habitual abortion* is based on the physiology of pregnancy. ENOVID provides balanced hormone support of the endometrium, permitting continuation of pregnancy when endogenous support is otherwise inadequate.

**6. Endocrine infertility.** ENOVID has been used successfully in *cyclic* therapy of endocrine infertility, promoting subsequent pregnancy through a probable "rebound" phenomenon.

## THE BASIC DOSAGE

Basic dosage of ENOVID is 5 mg. daily in *cyclic* therapy, beginning on day 5 through day 24 (20 daily doses). Higher doses may be used with complete safety to prevent or control occasional "spotting" or breakthrough bleeding during ENOVID therapy, or for rapid effect in the *emergency* treatment of dysfunctional uterine bleeding and threatened abortion.

ENOVID is available in tablets of 5 mg. and 10 mg. Literature and references, covering more than six years of intensive clinical study, available on request.

SEARLE

*Research in the Service of Medicine*

1962 ANNUAL SESSION — *Continued from Page 230***REPORT BY DOCTOR HAROLD N. WILLARD REGARDING PILOT PROJECT IN NURSING HOMES IN WATERVILLE**

The presentation of this report brought on discussion regarding the Kerr-Mills Bill and it was

VOTED that the committee appointed to study the implementation of the Kerr-Mills Bill, "study all of these things" and report back to the council.

This committee consists of:

Philip P. Thompson, Jr., M.D., Portland, Chairman

Carl E. Richards, M.D., Sanford

Harold N. Willard, M.D., Waterville

**KING-ANDERSON BILL:** A motion was presented by Doctor Carl E. Richards of Sanford "that the House of Delegates of the Maine Medical Association go on record as opposing any form of medical care for the aged under the Social Security approach, and that they reaffirm their support of the Kerr-Mills Bill, now being implemented within this State." Following considerable discussion, a roll call vote by counties was taken. Result — the motion was carried by a 23 to 22 vote.

(At the general assembly on Monday, June 18, it was voted unanimously "that the Maine Medical Association go on record as being against the King-Anderson bill.")

**AMPAC:** Doctor Richards reported briefly concerning AMPAC and MEMPAC. He stated that pamphlets relative to AMPAC and MEMPAC have been mailed to all members of the Association.

**REPORT BY DOCTOR ROBINSON L. BIDWELL OF PORTLAND, CHAIRMAN OF A COMMITTEE APPOINTED TO REVIEW THE SIZE, MAKE-UP AND MODE OF OPERATION OF THE COUNCIL**

VOTED that this report be referred back to the local county societies with instructions that it be given to the county delegates for action to be taken at the interim meeting in April, 1963. (Copy of this report has been sent to the County Secretaries.)

**MEDICAL ADVISORY COMMITTEE:** Doctor Thomas A. Martin of Portland, Chairman. Doctor Martin's report was a resumé of cases during a part of 1961 and up-to-date. He stated that in classification "A" money had been paid out on twelve cases totalling \$33,925.00, or \$2,827.00 per case. Files have been closed and claims abandoned on seven cases and at the present time, six cases are in suit and eleven cases are "open."

In reference to expenditures in class "A" he stated that "I think if we can buy those cases for that figure, compared to some of the other states, we did well, but we still don't like that much against us . . . The average for the previous year was \$3,777.61."

**HEALTH INSURANCE COMMITTEE:** Doctor Francis A. Winchenbach of Bath, Chairman. "Mr. Speaker and members of the House of Delegates. I think it is about time the Association was told that they are pretty good fellows. The Health Insurance Committee is an excellent committee, and they do work. When we have a meeting, everybody is there, even the fellows from up country.

"This committee, as you know, reviews all the claims from the insurance carriers, other than Blue Cross-Blue Shield. We have some complaints there, but not many.

"The boys, in general, are doing very well. We have a few we have to question once in a while. There were a couple who were sent to the Committee on Ethics and Discipline, for a little review, and such a review always helps. . . .

"I might say that we had a few variances in charges. You know, it is amazing, the range of the fees on some things.

"We did approve this over-age program; as you know, that was to be a national program, with the National Blue Shield Association, and it was to have gone into a pooled fund. We went into all of that. But that no longer exists. Now, it is to be administered by the state-local plan.

"At our last meeting, we voted to take no action as yet, and to recommend that you take no action as yet, in the local operation of an over sixty-five program, and we are awaiting developments from other states. . . .

"At the moment, we ask that you just abide by the committee's decision, and we will await results, as to what we can put into operation at a later date. . . .

"The Medicare contract has been renewed for 1962-63. There were some minor recommendations that the Committee approved.

"I wish to thank the Committee for their great support. You should be proud of them."

**NOMINATING COMMITTEE:** The report of the Nominating Committee, consisting of Standing Committees for 1962-1963 and for a delegate and alternate delegate to A.M.A. to serve from January 1, 1963 to January 1, 1965, was accepted. This report was published in the July, 1962 issue of The Journal of the Maine Medical Association — page 174.

**REPORT OF WOMAN'S AUXILIARY TO MAINE MEDICAL ASSOCIATION:** Mrs. S. Dunton Drummond, Bar Mills, President. Mrs. Drummond stated that the Auxiliary was formed to assist our "busy physician husbands. Sometimes, we feel that the Auxiliary is not needed. However, when we were asked to assist the Maine Medical Education Foundation, every one got to work and started to raise money." She said that their cookbook, "Rx 3 Times a Day" is a best seller and presented Doctor James A. MacDougall with a check for \$200 for M.M.E.F. (\$1,000 was presented to the fund at the annual meeting of the Auxiliary in May).

**PRESENTATION OF ROBINS AWARD** to Doctor George J. Robertson of Waterville for community service during the past year.

**PRESENTATION OF PAUL REVERE BOWL** to Miss Madeline A. May of Boston, stenotypist, who has served the Association in this capacity for twenty-five years.

**FUTURE MEETINGS OF THE HOUSE OF DELEGATES.** The Speaker of the House, Doctor Stitham, at the close of the afternoon session said that "we obviously are not giving the proper amount of time to the business at hand" and urged that some thought be given during the next year to the advisability of lengthening the time of this meeting, "so that we may expedite our work, and still give our members time for a little pleasure."

The complete report of the meetings of the House of Delegates is on file at the Association's headquarters and is available to any member of the Association.

# Maine Cancer Society†

Following is a partial list of films for use by members of the medical profession which are available on loan by request to The Maine Cancer Society, Federal and Green Streets, Brunswick, Maine as is information relative to films not included in this list.

## Motion Pictures

- After Mastectomy, 16mm, color (20-minute) (Oregon Division Film)
- Exfoliative Cytologic Method in the Diagnosis of Gastric Cancer, 16mm, color (22-minute)
- What Is Cancer?, 16mm, color (25-minute)\*
- Routine Pelvic Examination and Cytologic Method, 16mm, color (14-minute)

## The Problem of Early Diagnosis Series (16mm, color)

- |                                      |                             |
|--------------------------------------|-----------------------------|
| Breast Cancer (34-minute)*           | Oral Cancer (33-minute)*    |
| Cancer (30-minute)*                  | Lung Cancer (28-minute)*    |
| Gastrointestinal Cancer (33-minute)* | Uterine Cancer (21-minute)* |

## Kinescopes (16mm, color) — Physicians' Conference on Cancer

- Cancer of the Colon and Rectum (39-min., 1 reel)
- Cancer Detection (38-min., 1 reel)
- Chemotherapy: A Research Frontier (44-min., 1 reel)
- Diagnosis of Breast Cancer (45-min., 1 reel)
- Differential Diagnosis of Uterine Bleeding (45-min., 1 reel)
- Head and Neck Cancer (45-min., 1 reel)
- Lymphomas and Leukemias (55-min., 2 reels)
- Management of Advanced Cancer (46-min., 2 reels)
- Psychological Aspects of Cancer (39-min., 1 reel)
- Tumors of Childhood (44-min., 1 reel)

\*Replacement Leader and Title Section

†Submitted by the Professional Education Committee of the Maine Cancer Society, Eugene E. O'Donnell, M.D., Chairman.

A new film for the medical profession, **CANCER DETECTION: PROCTOSIGMOIDOSCOPY IN OFFICE PRACTICE**, has been prepared by Dr. Emerson Day in cooperation with the Strang Cancer Prevention Clinic and Memorial Hospital for Cancer and Allied Diseases, New York City.

This 15-minute, 16mm., color-sound film and a teaching booklet are available without charge upon request from Modern Talking Pictures Service, Inc., 3 East 54th Street, New York 22, N. Y.



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### Eighth Grade Tuberculin Skintesting

School Year 1961-1962\*

KATHARINE D. GAY\*\*

The United States Public Health Service gives the national average tuberculin reactor rate for fourteen year olds, at the present time, as 3%. The prevalence of tuberculin sensitivity among eighth grade students (thirteen and fourteen year olds) in Maine at the present time is 2%. It is the aim of those working in the tuberculosis field to reduce the tuberculin reactor rate in Maine among eighth grade students to 1% by 1970.

The present reactor rate of 2% in the State of Maine, among eighth grade students, was determined during the school year 1961-62 as a result of the following described conference and project:

In April 1960, following the pattern of the National Tuberculosis Association and the United States Public Health Service, who had held a joint meeting some months before at Arden House, in Harriman, New York, a similar meeting was held in Maine. The Maine meeting was jointly sponsored by the Maine Tuberculosis and Health Association, the Maine Medical Association, and the Department of Health and Welfare.

The purpose of the meetings was, respectively, to survey the total tuberculosis situation throughout the nation and the State of Maine, and to propose both immediate and long range goals for the eventual eradication of the disease.

Statistical information presented on nationwide and statewide bases indicated that while substantial progress in diminishing both the case and death rates had resulted from past efforts in control, there still remained

pockets of comparatively heavy infection, which if not controlled, might serve as sources of future widespread infection. Therefore, an all out attack, aimed at elimination rather than control, was considered to be justified, and certain immediate and long range goals were established as guide lines.

One of the more immediate goals on a nationwide basis was the reduction of the tuberculin skin reactor rate among fourteen year old children to not over 1%. It was stated that such a reactor rate would indicate good control of the spread of infection in any community.

Although many pupils in the eighth grade are only thirteen years of age it was decided to determine the reactor rate in Maine among eighth grade school children rather than confine it to fourteen year olds only. The reason for making this choice was that in most Maine communities the school system is based on eight elementary grades, and four high school grades. It was felt that the project could be carried on easier in the elementary school system than in high schools.

As a first step in the project the Division of Research and Vital Records of the Department of Health and Welfare was asked to submit a random sampling of towns, covering all six health districts of the State, which would produce approximately three thousand school children in this school grade. It was the opinion of the Director of the Division of Research and Vital Records that this number of children would represent an adequate sampling.

The project was carried on under the supervision of the respective State district health officers. Permission for testing in the schools, and preliminary classroom educational activities were obtained from superintendents and principals by the field workers of the Maine Tuberculosis and Health Association. In some districts

\* A study conducted in a random sampling of towns throughout the State.

\*\* Administrative Assistant, Division of Tuberculosis Control Department of Health and Welfare, Augusta, Maine.

they also assisted the physician at the testing and reading sessions. In some of the health districts the district health officer himself did the testing and reading of the reactions; in others the district health officer arranged with the local school physician to test and read reactions.

All necessary forms, PPD, and syringes were supplied by the Department of Health and Welfare. The assembling of the statistical results was also a responsibility of this department.

The intradermal method of testing was used in all instances. Intermediate strength PPD (5TU) was used. Reactions where induration measured 5 mm and over were designated as positive.

All positive reactors were given a free 14x17 chest film. A total of 3,630 children were tested, both male and female. A total of 72 reacted to the test.

The percent of reactors varied from district to district and from town to town. The overall reactor rate was 2% (1.99%) or practically twice the percentage stated that would indicate good control of tuberculosis.

By health district, and towns chosen, the percent of positive reactors was as follows:

DISTRICT I				
Town	Percent of Participation	No. Tested	No. Reactors	Percent of Reactors
Westbrook	93	262	6	2.29
Waterboro	96	22	0	—
Gray	83	49	0	—
Biddeford	84	318	3	0.95
Elliot	82	55	0	—
Shapleigh	100	9	0	—
District Total	88	715	9	1.24

DISTRICT II				
Town	Percent of Participation	No. Tested	No. Reactors	Percent of Reactors
Durham	100	28	0	—
Hartford	100	5	0	—
Mexico	95	110	0	—
Lewiston	92	727	21	2.88
District Total	94	870	21	2.42

DISTRICT III				
Town	Percent of Participation	No. Tested	No. Reactors	Percent of Reactors
Fairfield	91	126	5	3.97
Rockland	99	151	6	3.96
Warren	100	38	0	—
Monroe	100	10	0	—
District Total	96	325	11	3.38

DISTRICT IV				
Town	Percent of Participation	No. Tested	No. Reactors	Percent of Reactors
Bangor	92	633	14	2.22
Brownville	93	37	0	—
Milford	89	24	0	—
Millinocket	99	171	1	0.59
Greenville	94	62	0	—
Brewer	96	168	4	2.38
District Total	94	1,095	19	1.73

DISTRICT V				
Town	Percent of Participation	No. Tested	No. Reactors	Percent of Reactors
Mt. Desert	93	26	1	3.84
Bucksport	96	81	2	2.47
Bluehill	100	28	0	—
District Total	97	135	3	2.22

DISTRICT VI				
Town	Percent of Participation	No. Tested	No. Reactors	Percent of Reactors
Ft. Fairfield	100	147	4	2.72
Limestone	96	55	2	3.64
Sherman Mills	86	25	0	—
Caribou	93	263	2	0.76
District Total	95	490	8	1.63

The number of children involved in the project in many of the towns was very small. In such instances a single reactor gave a high reactor rate. For example a single reactor reported from the town of Mt. Desert gave that town one of the highest rates in the group, i.e. 3.84%.

Study of the preceding figures indicate that children in the larger population areas react positively to a tuberculin skin test more frequently than those who live in the more rural areas.

In order to reduce to 1% the tuberculin sensitivity rate among eighth grade school children in the State of Maine by 1970 the following phases of the total program need strengthening:

1. Discovery and adequate treatment of the presently undiagnosed case
2. Adequate treatment of the already diagnosed case
3. Vigilant follow-up of the inactive case to avoid reactivation
4. Adequate follow-up of tuberculin reactors
5. Prophylactic treatment of recent tuberculin converters to prevent the initial occurrence of clinically active disease among this group.

## Annual Influenza Vaccination As A Lifesaving Measure\*

"... the large influenza pandemics of 1918-1919 and 1957-1958 caused the death of millions of persons throughout the world. It has not been so well appreciated that excess mortality directly associated with outbreaks or epidemics of . . . influenza virus can be shown in many of the intervening years. . . . More than half of these excess deaths occurred in persons with cardiovascular-renal disease and over two-thirds of the total were in those who were 65 years of age and older.

"Clinical studies have documented the association of rheumatic heart disease and influenza associated deaths. The occurrence of influenza virus pneumonia in patients with mitral stenosis has been frequently noted. . . . the dangers of influenza to pregnant women have been the subject of clinical observations that have described increased number of fatal pneumonias during epidemics. . . . Other conditions that place a patient in the high risk group include chronic pulmonary disease and metabolic disorders such as diabetes mellitus.

"Patients in the following disease categories have experienced the highest mortality rates and, therefore, specific protection is clearly indicated for them as a routine practice:

- A. Persons at all ages who suffer from chronic debilitating diseases such as chronic cardiovascular, pulmonary, renal, or metabolic disorders; in particular
  - 1. Patients with rheumatic heart disease, especially those with mitral stenosis.
  - 2. Patients with other cardiovascular disorders, such as arteriosclerotic heart disease and hypertension, especially those with evidence of frank or incipient cardiac insufficiency;
  - 3. Patients with chronic bronchopulmonary disease, for example, chronic asthma, chronic bronchitis, bronchiectasis, pulmonary fibrosis, pulmonary emphysema, pulmonary tuberculosis;
  - 4. Patients with diabetes mellitus;
  - 5. Patients with Addison's disease.
- B. Pregnant women.
- C. All persons over 65 years of age."

(Mogabgab, William J., American Heart Journal, Volume 62, pages 587-589, 1961.)

\*Submitted by the Maine Heart Association, Inc.



# The Journal of the Maine Medical Association

Volume Fifty-Three

Brunswick, Maine, October, 1962

No. 10

## Laryngostomy For Acute Airway Obstruction

LORING W. PRATT, M.D.\*

Tracheostomy is frequently a life-saving procedure and all physicians should be acquainted with the quickest and the most practical way of handling it in an emergency. For those situations in which time is of the essence, equipment is in short supply, or such a life-saving technique must be utilized under trying circumstances, laryngostomy via the cricothyroid membrane is clearly indicated.

An opening is made through the skin over the cricothyroid membrane into the subglottic larynx and an airway is established at this level. This provides a satisfactory airway only for short-term use and it should be closed at the early convenience of the physician after a classical tracheostomy has been established under ideal circumstances.

### PROCEDURE

A vertical mid-line skin incision is made from the notch of the thyroid cartilage to the upper part of the trachea. This is carried down to the perichondrium of the thyroid and cricoid cartilages and, in the area between these two cartilages, the cricothyroid membrane is exposed. The skin is retracted and a transverse incision made through the cricothyroid membrane into the lumen of the larynx. A tracheostomy tube, or some substitute, is then inserted through this incision and the airway is established. (Fig. 1 and 2)

The entire procedure can be completed in a few brief moments and this is its great value. At this level the airway is very close to the skin, there being in many patients not much more than a quarter of an inch be-

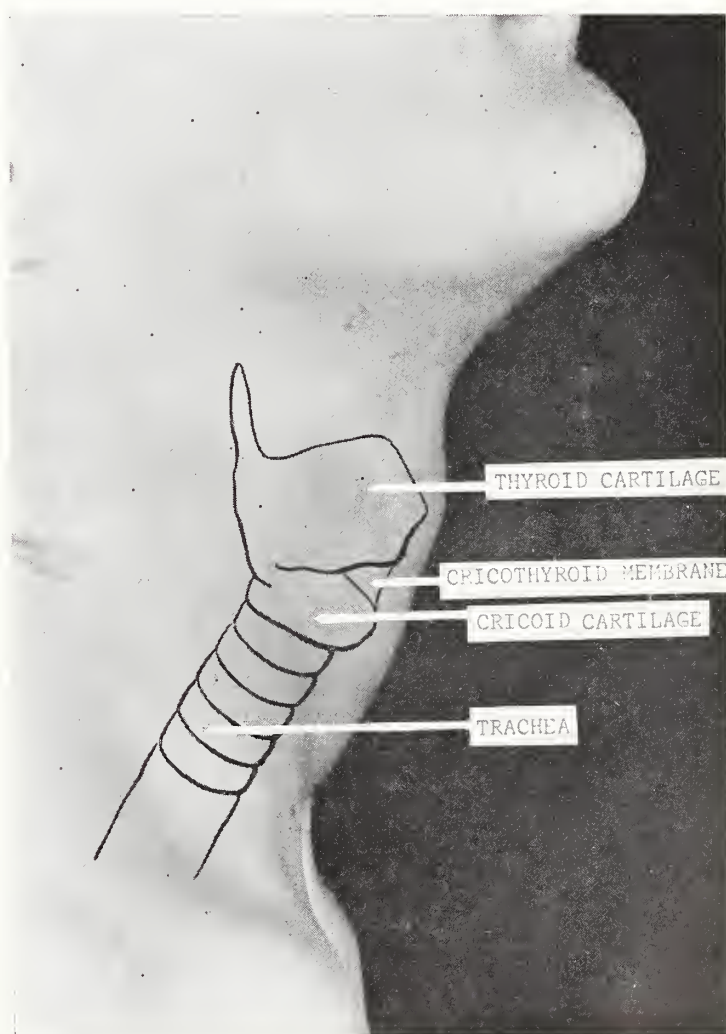


FIG. 1. Lateral view of slim female neck showing position of larynx and trachea and relation of cricothyroid membrane to normal landmarks.

\*Professional Building, Waterville, Maine

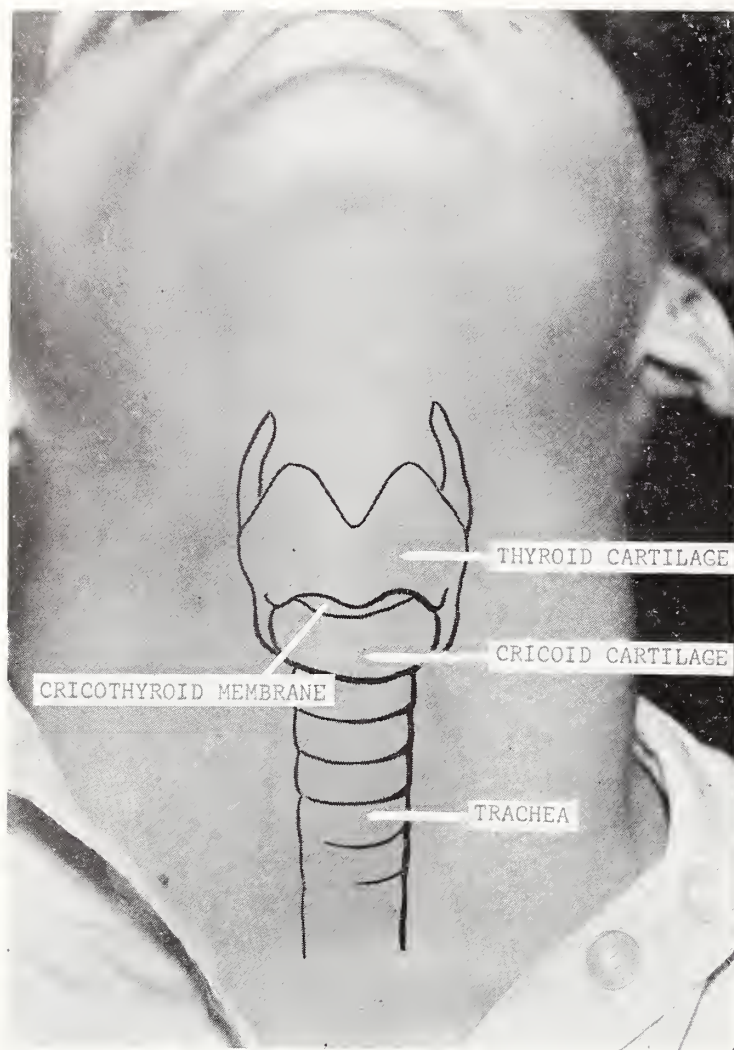


FIG. 2. Anterior view of slim female neck showing position of larynx and trachea and relation of cricothyroid membrane to normal landmarks.

tween the surface of the skin and the lumen of the airway.

The operation can be carried out by the use of a knife alone. Retractors are not necessary although they would undoubtedly be helpful. As deep dissection is

unnecessary and no major vessels are found in the midline in this area, it is unlikely that any troublesome bleeding will be encountered. This technique provides an entrance to the airway below the level of the vocal cords, hence the obstruction of a foreign body, edematous epiglottis, edema of the vocal cords or other fixed obstruction of the upper airway is circumvented. Access to the lower airway for suction to relieve fluid obstruction is also obtained.

The chief danger involved in this procedure is that damage may be caused to the cricoid cartilage. As this cartilage forms the only complete ring of the laryngeal cartilages its integrity is essential to the preservation of a laryngeal lumen. Such damage may result in laryngeal stenosis. However, in acute urgent situations, even damage to the cricoid cartilage would be less catastrophic than death. For this reason laryngostomy is a highly desirable procedure in many circumstances.

#### CONCLUSION

1. All physicians should be proficient in the surgical relief of acute obstructive respiratory distress.
2. In urgent situations, in situations where surgical equipment is in short supply or lacking, and in situations where the surroundings are not conducive to good surgical technique, it is suggested that laryngostomy may be useful.
3. Laryngostomy is a simpler, quicker procedure than tracheostomy and is equally efficacious in providing an airway.
4. Maintenance of a laryngostomy is not desirable over a long period of time and should be closed at a later hour after an elective low tracheostomy has been established.

#### ACKNOWLEDGMENT

I would like to acknowledge the artistic work done by Miss Jean Ann Pollard in the preparation of this paper.

#### A Fine Balance In Drug Legislation

It is of fundamental importance that new drugs, before being used widely in research and being marketed, should be adequately tested for safety. Of equal importance is the fact that there be a continuous flow of new and improved drugs. Any legislation on this subject, therefore, needs to strike a fine balance. On the one hand, it properly seeks to broaden and strengthen controls over drugs both new and old, to protect the public interest. On the other hand, it must encourage rather than obstruct the continuing flow in the number and kind of new drugs that are needed for better health. — Leonard A. Scheele, M.D., Senior Vice President, Warner-Lambert Pharmaceutical Company, to House Interstate and Foreign Commerce Committee, August 20, 1962.

# Medical Obscurities Of The Civil War

JAMES E. POULIN, M.D.

In this centennial year of our historic Civil War, publications throughout America are abundant with articles, and stories pertaining to battles, leaders, and the part they played in this conflict. A review of this literature discloses little if any mention devoted to the role that medicine and surgery played in this critical struggle between the states. A study of the writings of that period reveals that much was written on the subject and many little-known medical facts were discussed. It might be of interest to present in this paper some of the pertinent complex problems that faced medical men of that period, one hundred years ago.

At the outbreak of the war, the United States Surgeon-General's office consisted of a total of one hundred and fifteen surgeons. Twenty-four of these resigned to form a nucleus of the Confederate medical service. Eventually both services were vastly though inadequately expanded. According to present medical standards this was, a very meager number. Nursing services were equally primitive. The army relied on male nurses, most of whom were untrained and unfit to take care of desperately-ill soldiers. We must remember when we read of the works of the surgeons and their high mortality, that antiseptics were unknown, that the relation of dirt to infection was not generally understood, that anesthesia was just coming into general use, and that drugs were most inadequate. It is not surprising then, in the light of all this, that mortality from disease and wounds was far greater than from bullets and that hospitalization was also regarded as an equivalent to a death sentence. I have been unable to find accurate statistics, but it is a safe generalization from reports available that lingering deaths from wounds were as numerous as sudden deaths on the battlefield, and that deaths from disease were far more than twice both of these combined.

The war between the states was fought in the very last years of the medical middle ages. It was a period of crudeness in medicine and surgery. While the guns were firing throughout the South, Pasteur, in France, was laying the groundwork for bacteriology. Within two years after the last battle of the Civil War, Lister was beginning the application of his aseptic method. Recovery from wounds or disease in this dark period of medicine was infinitely more difficult than today because the physician had a very limited supply of commonplace drugs, none of which were endowed with antibiotic powers. The surgeon of that time did not have an organization to direct him or supply him with the necessary instruments and drugs such as is done today. It was necessary that he be extremely re-

sourceful and carry out his work without the use of chloroform or other anesthetics that were available even in those days. The ambulances used by both sides to carry the wounded to places of safety or railroad terminals were generally very poor; for the most part they were without springs and certainly unfit for a wounded soldier to be carried upon. Records indicate that wounded soldiers, if at all able, would prefer to walk to the regimental hospitals than to go by ambulances, for ambulance rides might well induce sufficient trauma to open up the wound with subsequent fatal hemorrhage. Jarring rides from the battlefield in one of these two-wheeled ambulances often proved fatal to the badly wounded. A converted passenger coach served as a Federal Hospital car holding thirty-three wounded. Often, the seats were removed and replaced with wooden slats. The least seriously wounded rode in stretchers hung from above.

It is difficult to get an accurate count of numbers who served in Civil War armies. The Union had the equivalent of about one million, five hundred thousand three-year enlistments from the first to the last, and the Confederates had about 1,000,000 in all. Approximately 359,000 Federal soldiers and 258,000 Confederate soldiers lost their lives in the cause of the war. These figures, to be sure, include death from disease as well as battle casualties, but a young man who died of dysentery was just as dead as one who stopped a bullet, and when these figures are matched against the total possible enrollment, they are appalling. The unfortunate Civil War soldier, whether he came from the North or from the South, entered the service when the killing power of weapons was approaching a new peak of efficiency. It was also the closing years of an era when the science of medicine was woefully, incredibly imperfect, so that he got the worst of it in two ways. When he went forth to battle, he was likely to be hurt pretty badly; when he stayed in camp, he lived under conditions that were very likely to make him sick; and in either case he had almost no chance to get the kind of medical treatment which, a generation or so later, would be routine. Both the Federal and the Confederate governments did their very best to provide proper medical care for their soldiers, but even the best was not very good. This was nobody's fault. There simply was no such thing as good medical care in that age, not, at least, as we understand medical care today.

It is interesting to note that some gunshot wounds of the abdomen, particularly those penetrating a viscus, were regarded as almost certainly fatal. Except when it was necessary to return the protruding viscera, no

abdominal operations were attempted. It was believed that when this important cavity was once penetrated, death was inevitable. In such cases the surgeon did little more than attempt to soothe and to relieve the patient by administration of opium. Wounds of the chest were regarded as hardly less serious. If the patient was sufficiently strong, blood was drawn from a large vein until the patient fainted. Towards the latter part of the war, however, the status of bleeding became dubious and was used less frequently. Wounds of the head and neck constituted about 12% of the total wounds. This was the chief field for surgical intervention during the period of the Civil War.

The one and notable surgical procedure of the Civil War period was surgical amputation. The extent to which this mutilating operation was resorted to is appalling. An early surgical dictum was that practically all gunshot wounds of the femur and penetrating wounds of the joints were fatal, and immediate amputation offered the only opportunity for recovery. The mortality statistics of this particular procedure are interesting. Amputation of the leg was associated with a mortality of approximately 43%; of the thigh, with a mortality of 49%; of the forearm with a mortality of 13%; of the arm, with a mortality of 28%. Gunshot wounds of the thigh accompanied by compound fractures were treated with amputation with a mortality of 60%. When one views these tragic figures, it is easily understood why the general overall mortality in the Civil War was so high and infection controlled the fate of all soldiers.

Chloroform was the anesthesia of choice in both armies. In addition, ether was used in the North and was preferred by the Union command. The most amazing fact is that although these anesthetics were used, hundreds of thousands of times in this Civil War, there were only 37 reported deaths from anesthetics. This is most remarkable when one considers the very crude manner in which they were administered both in the hospitals and on the field. Surgeons of the period believed that suppuration associated with wounds was a normal and a necessary part of the tissue repair; they were astonished when an occasional wound healed without it.

Tetanus was recognized as a disease, but hardly understood at the time; it was regarded as a rare complication with no specific treatment. Without a doubt its rarity was due to the fact that it was seldom diagnosed correctly. Prior to all surgery, brandy was the premedication of choice. The Confederate archives speak of the desperate shortage of medical supplies and instruments which resulted from the Union blockade and this necessitated heroic improvising. It was necessary for the medical department of the South to seek in the forests for substitutes or drugs that could not be obtained from the North. Poppy fields in Florida and North Carolina were commonplace to supply the opium that could not be imported. In spite of the blockade, chloroform, quinine and opium were always

available in the medical department; this fact is not understood by modern historians.

Antiseptics and asepsis were not understood at the time of the war between the states. The discovery of Lister came ironically just two years after the war ended in 1867. Instruments were always unsterile, but southern surgeons cleansed their wounds with rags that had been washed clean, boiled and ironed. It was intended as an economy measure without any thought of sepsis. This was in contradistinction to the care of the wounds of Union soldiers. Northern surgeons washed all wounds with an unclean sea sponge which was used indiscriminately in all cases. Sponges were not available to the South because of the blockade. The same sponges were used to wash the wounds of hundreds of injured and the sponges were washed usually in the same pail of dirty, disease-filled water. This bit of improvising by the surgeons of the south undoubtedly reduced their morbidity and perhaps their mortality. Blood vessels were ligated by northern surgeons with unsterile silk, the ends of which were left long and led out of the wound. When the ligatures became loose, because of autolysis of tissue, they would pull out, often with accompanying severe hemorrhages. The unsterile ligatures served as a capillary wick which allowed additional bacteria to enter the depth of the wound. It was indeed a very rare wound that was not complicated by some form of severe infection. In contrast to this, the South had no silk ligatures or sutures and they had to resort to horsehair which was boiled to make it more pliable; hence, by happy accident, the material buried in the wound was rendered sterile. Once again, without realizing it, improvising helped to reduce the mortality on the Southern battlefield. In all military hospitals erysipelas was a fearful disease. At that time it was called hospital gangrene. Mortality associated with it was very high. In the Southern hospitals, erysipelaslike wounds were washed three or four times daily with chlorinated water which was not unlike Dakin's solution. This was not done as an antiseptic irrigation, but rather to keep the odor down; without realizing it, this preparation may have saved many lives.

A substantial percentage of the injuries of battle were brutal wounds caused by the Minie ball, a conical rifle bullet named after its inventor. Its low velocity accounted for its destructive qualities which could produce horrible wounds. With low velocity, the Minie ball would turn in its course causing a bursting type of wound of exit. These wounds were much more severe than the wounds caused by the higher velocity bullets used by the sharpshooters. Strangely enough the wounds from cannons were very rare and wounds from sabres were extremely rare. The presence of a foreign body in the form of a bullet was regarded as an emergency and its extraction was carried out at once. Probing with unsterile, crude instruments frequently brought about hemorrhage and death of a patient who

might have lived had the bullet been allowed to remain in place. Hemorrhage was the prime cause of mortality and records disclose that three-fourths of those who died in battle perished from hemorrhage alone. Mortality from hemorrhage resulted from a lack of surgical skill, crude probing of the wound, and the rough ride in the ambulance to the rear. Infected wounds also frequently caused a large slough which produced massive, uncontrollable hemorrhages. Treatment of hemorrhage itself was provisional, inadequate and extremely conservative. It frequently consisted of pressure, elevation, application of cold, sedation and local styptics. Seldom was the wound enlarged so that suture or clamp could be applied to the bleeding vessel. Of the wounded soldiers who lived to reach a hospital, 70% had wounds of the extremities. It was known to all surgeons of the period that a gunshot wound into a joint or a gunshot fracture of the femur would nearly always be fatal if not operated upon. Statistics available reveal that 90% of the patients with compound fracture of the femur would die if the limb was not amputated. With amputation the figure was lowered to about 60%. It is further recorded that delayed surgery or secondary operations carried a mortality which was approximately double that of the primary operation. Thus for operative procedures such as an amputation of the femur, the mortality was 30% if the operation was primary; while if the operation were delayed or secondary, the mortality was 78%. The mortality for the amputation of the arm was 14%, while the mortality for delayed surgery of the same procedure was 37%. Approximately 20% of the wounds incurred were of the torso. Abdominal wounds which perforated a hollow viscus were almost uniformly fatal. Although the pathologic physiology of sucking chest wounds was understood imperfectly, medical men at that time suggested that gunshot wounds of the chest be treated by sealing the wounds of entrance and exit. This prevented the additional entry of air into the pleural cavity which would collapse the lung and cause a mediastinal shift. Approximately 10% of the wounds of battle involved the head and neck. Fractures of the mandible were treated in some of the armies by the application of a cardboard splint which was padded with cotton and secured by a bandage with a double tail attachment.

As staggering as casualty figures for Civil War battles are, even more appalling are the statistics of deaths from disease. It has been estimated that two and one half Union deaths resulted from disease for every single combat loss, while the ratio on the Confederate side was three to one. The North Carolina soldier who wrote that "these big battles is not as bad as the fever" knew of what he told.

The U. S. Sanitary Commission set themselves the task of raising living standards in camps and hospitals. Yet, despite these efforts, hospitals remained fearful places, and doctors were generally distrusted. "I insisted on taking the field. . . ." wrote an Ohioan ordered to

the hospital, "thinking that I had better die by rebel bullets than Union Quakery."

As in most wars, sickness was far more disturbing than wounds. In the hospitals both in the North and South, two-thirds of the admissions were for specific diseases rather than from battle wounds. Among the disabling diseases of the war, malaria ranked among the first, accounting for one-fourth of all the reported cases of diseases in the Union army. The disease was more prevalent among the Southern troops but less fatal than among the Northern troops, probably due to the Southerners' previous experience with the disease. Quinine had been the specific drug of choice in the South for several years, but quinine was an imported drug and the supply situation went from bad to worse during the war. Southern medical service used quinine as a prophylactic measure against malaria, but the scarcity of the drug prohibited this. This was not done by the Northern medical department. Typhoid fever, although less frequent, was responsible for about one-fourth of all the deaths and diseased in the Southern army. Later in the conflict, typhoid declined in both armies. Pulmonary tuberculosis as a disabling disease was probably far more common than the few cases which were reported by both sides. It is quite probable that its exact diagnosis was not established and that these patients died of pneumonia. Meningococcus meningitis was a rare disease among the troops on both sides. There are very few cases reported of its existence. The eruptive fevers with high incidence and appreciable mortality showed serious outbreaks in both armies. The fact that an overwhelming majority of the troops on both sides, especially those from southern rural areas, had little experience with the usual exanthematous diseases of childhood allowed them to fall easy prey to these diseases in the crowded army environment.

As everyone knows, vaccination was introduced into America in 1800, and it is with some surprise that smallpox was an active menace some sixty years later in both armies. This condition was due, not so much to leaving men unvaccinated as to bad vaccinating techniques and impure vaccines. The usual procedure was to use a crust of one vaccination as vaccine for as many as two hundred vaccinations. It became a fad in the army, despite a violation of orders, for soldiers to vaccinate one another using rusty knives and whatever instruments might be at hand. As a result of this practice, several thousands of deaths resulted from smallpox on both sides. This occurred sixty years after vaccination had been successfully carried out for smallpox. Measles played havoc with the troops on both sides, especially those enlisted men who came from rural areas and were not exposed to measles as men were from urban communities. One out of every seven men in certain divisions was stricken with this disease. For some strange reason, scarlet fever was rarely seen during the war, and mumps was recorded only during the first year of the conflict. Scurvy did exist, however, and

this occurred more often in the Southern armies. This, of course, is easily understood when one considers the scant rations of the Southern troops. Many of the Southern troops were obviously affected with what we know as night blindness which was certainly due to the lack of vitamin A in the diet. Dysentery was prevalent on both sides and it caused many men to be disabled. The cause at that time was not properly understood. Sanitation at that period of our national history was very poorly managed. As the war went on, observation taught the medical men that there was a positive correlation between the sanitation of the camp and its intestinal disease rate. Mortality associated with some of the dysentery and typhoid fever was extremely high. In some camps it was as high as 10%. In all army camps, diseases like typhoid and dysentery were dreaded killers. No one knew what caused them and no one could do much for them when they appeared. Doctors had discovered that there was some connection between the cleanliness of the camp and the number of men on sick call, but sanitation was still a rudimentary science, and if a water supply was not visibly defouled or odorous, it was thought to be perfectly safe. The intestinal maladies which took so heavy a toll were believed due to miasmatic odors or to even more subtle emanations in the air. This type of camp illness was the most difficult of all army diseases to cope with and the number of cases progressively increased as the war continued. It destroyed more soldiers than gunshot wounds. Also more soldiers were permanently disabled and lost in the service from these diseases than from the disability following accidents of battle.

So the soldier of the 1860's had everything working against him. In his favor, however, there was a great deal of native toughness, and a sardonic humor that came to his rescue when things in the camp were darkest; these, and an intense devotion to the cause he was serving.

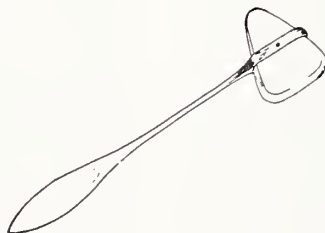
In spite of the magnitude of the war between the States it did not eliminate the doctor as an individual. Many country physicians who would have been destined to obscurity, in ordinary times, became national figures because of their war effort. Physicians were forced to deal with crises which called for great resourcefulness

and skill than was ever shown in peacetime. Some of the most interesting accounts of the war came to us from the pens of medical men. In spite of the crudeness of medicine in this dark period of American history, the vast experience gained on both sides served to widen the horizon of medical and surgical knowledge. Many small town surgeons who served developed initiative and enterprise that enabled them to become leaders in the medical profession following the war. A large percentage became professors of medicine and surgery in the medical schools throughout America. They assisted in the establishment of new hospitals, and they introduced medical standards that might not have occurred in the next decade. Textbooks were written that stood the test of time for the next fifty years. The surgeons of this desperate struggle did not achieve the immortality of Lee or Grant, but they did more to advance the cause of both sides, than any other group. The most tragic aspect of Civil War medicine is the fact that so many young men were lost during their most productive years to wounds and disease which would have been preventable or curable by our present-day knowledge. Even the death of the military heroes on both sides was due not to defective treatment, but to the meager facilities and the relative backward state of medical science at that time. The intelligent surgeon-generals and the loyal cooperation of their colleagues enabled the Southern resistance to carry on for four years. Simultaneously, Northern opposition could not have ended the conflict without its very capable medical leaders. It is my belief that the best surgeons in the world could be found in the ranks of the Northern and Southern armies at the conclusion of the Civil War.

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# Alcoholism — A Public Health Problem

RICHARD H. WHITTEMORE\*

The prevalence of *alcoholism* in the state of Maine is high enough to warrant attention as a serious *public health problem*. Most every town or city of any size in Maine has its "skid-row," "slum area" and "honkytonk joints," but these are not the cause, or always the end result of alcoholism, a disease that has no respect for persons, rich or poor, educated or underprivileged, men and women, young and old on every rung of our social structure.

"Alcoholics are those excessive drinkers whose dependence upon alcohol has attained such a degree that it shows a noticeable mental disturbance or an interference with their bodily or mental health, their interpersonal relations, and their smooth social and economic functioning; or who show the prodromal signs of such developments. They therefore require treatment." This definition was published by the World Health Organization, Alcoholism Sub-committee, in August 1952.

Various titles have been used to refer to those who have abnormal drinking patterns: *Excessive drinker*; *problem drinker*; *addictive drinker*; *uncontrolled drinker*; and *alcoholic*. There are many attitudes about "drinking," and each title projects a different mental picture, interpretation and attitude for each individual, depending upon his or her early training, experiences and relationships with "tea-totalers," "social drinkers" and "alcoholics," plus his or her personal experiences in the use of alcoholic beverages.

Dr. Seldon D. Bacon, Ph.D., Director of the Yale Center on Alcohol Studies, gives this non-technical definition for alcoholism: "Alcoholism is a medical and social disorder characterized by the uncontrolled use of alcohol and the progressive disorganization of the physical, psychological and environmental effectiveness of an individual."

Recent figures indicate, using the above definitions, that there are five million *alcoholics* (male and female adults) in the United States today. Using the same formula we arrive at approximately 27,000 in the State of Maine and still on the increase. If these figures referred to polio or small pox we would be well aware of a serious epidemic. As far as we know *alcoholism* is not contagious, but alcoholism has been termed a *disease* by sociologists, psychologists and medical authorities.

Dr. Marvin A. Block, M.D., Chairman, Committee on Alcoholism, American Medical Association Council on Mental Health writes: "With the new positive approach to this public health problem, the physician

treats these sufferers as sick people. He recognizes personality defects in those who react so unusually to alcohol. He knows that the problem drinker has an immaturely developed personality and uses alcohol to escape life's pressures and relief from the tensions which living often entails. *Such patients employ alcohol as a drug*. For many years doctors have studied the physical and emotional characteristics of this illness. Much has been learned, but as with many other medical problems, the specific answer has not yet been found."

## CAUSES

*Escape* is sometimes over-emphasized when explaining the causes of alcoholism. Could the alcoholic not be seeking something else . . . sociability, excitement, romance, adventure? Perhaps it could be a spiritual search? On the other hand a *social drinker*, who has nothing to escape from, but who enjoys the taste and effect of liquor, might become an alcoholic through continued, excessive use.

There is no positive way of identifying the "potential alcoholic." If anxiety, fear, tension and personality conflicts alone cause alcoholism, then over half the population of this country would be alcoholic. It takes a combination of many factors to produce alcoholism. It is a *total disease*, involving the whole person, mentally, physically, socially and spiritually.

The drinking of alcoholic beverages is socially and legally accepted in most parts of this country today. Therefore, most people take up drinking as an "accepted custom." They are not threatened with the possibility of becoming an alcoholic. This is always the least of their worries. The beginning is innocent enough. Smokers today can worry about cancer, but the drinker never worries about *alcoholism*. He doesn't worry about it even after problems begin to arise in connection with his (or her) drinking habits because the mental picture of the alcoholic is that of the "bum on skid-row," and no one wants to identify himself with this.

Causes? Who can say what the real causes are? They can be as many and as varied as there are alcoholics. There are, however, a few outstandingly noticeable symptoms: emotional immaturity; mental obsessions; physical compulsion, and an over-active conscience.

## SYMPTOMS

Most adverse situations can emotionally upset alcoholics to such an extent that they have to go out and throw their tantrums by getting drunk. On the other hand, if they get a raise in pay, win a bet, or if the wife

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has a baby, they must celebrate by drinking. (Usually, but not always intentionally, they get drunk.) They use liquor as a panacea as well as a narcotic.

The alcoholic has obsessions that drinking improves his personality; that he is a more interesting, intelligent and productive person "with a few drinks aboard." He usually feels dull and bored when sober. He believes he cannot have any fun at a social function until he is "feeling high." The alcoholic does not always drink to find the courage to perform immoral or illegal acts. He might, however, perform such acts while under the influence because of reduced controls.

We find that most alcoholics have a physical compulsion . . . a hunger, a thirst for more and more alcohol after they have taken the first drink. In some instances it could be a psychological desire to sooth a physiological pain . . . "raw nerves." It could be just the desire to hold onto a "warm comfortable feeling." This craving can in some instances be attributed to a faulty metabolism, or glandular disfunction.

"Observers now believe that no one particular phase can be held accountable (for alcoholism); that the total personality is involved physiologically and psychologically. As in most illnesses, the two phases work together," states Dr. Block.

Socially the alcoholic "fits" or he "doesn't fit," depending upon whether or not his "group" sanctions "excessive drinking and intoxication." If he "fits" he continues drinking openly with the group. If he is out of order, (drinks beyond the accepted norms) then he becomes belligerent and a problem to the group, or he will seek out a group where he "fits," or he will try to drop out of society entirely and become a "lone drinker." If his family will not tolerate his actions he will continue on as a "sneaky drinker." But regardless of the group he belongs to he is a problem to his family, his employer, and his community. For a long time this person is not aware of, nor does he admit that he has a *drinking problem*.

If the alcoholic has been raised in a "good home" he is going to be further tormented by an over-active conscience. Thoughts of "weakness" and "guilt," of unpleasant and embarrassing experiences are going to fill him with regret and remorse until he loses self-respect and confidence. Now the "pain" is so great that he must find relief. And where does the alcoholic go to find relief? Back to the bottle, in spite of previous promises and threats.

The alcoholic experiences *very real suffering*.

Until the attitudes of society change toward the *sickness of alcoholism*, everyone is going to suffer, either directly or indirectly. *The answer is:* more information through *education with facts* about alcohol and alcoholism. Only through mature understanding can we *prevent* and *treat* alcoholism. Although there is no known *cure* for alcoholism, *treatment* is necessary and can be effective in "arresting" the disease. The proof lies with the thousands of men and women living today who

have overcome their problem, who have recovered, and have returned to their normal place in society; who have learned that "as long as they don't take that first drink" their case is *arrested*. They must be motivated with a *stronger* desire *not to want to drink* than a desire *to drink*. This can be accomplished in many ways as will be explained later.

The treatment of alcoholics could go on indefinitely without gaining ground. Treatment is necessary, yes, but it is not the most important solution to this serious public health problem.

Only through the study and application of preventive methods have our public and private health agencies been able to solve such problems. How true the old saying: "An ounce of prevention is worth a pound of cure."

Methods for the prevention of alcoholism are still going through the early experimental stages in many parts of the world. At this point I would like to make a few personal observations and suggestions from my own experiences and study of the problem. These will be short and to the point: We must in some way get information about *alcohol* and *alcoholism* across to boys and girls in the 14 to 21 age group. *We must be uniform in what we teach*. We must stick to *facts* about alcohol and alcoholism, avoiding all "Wet vs Dry" issues, *leaving the moralistic aspects of the problem* to the families and to the individual's conscience to interpret. *With co-operative and coordinated support* this could be successfully carried out in the schools and churches, and in the doctor's office.

Young people must be informed of the facts: That alcohol is not a "stimulant," it is a "depressant" an anesthetic; that alcohol, circulating in the blood stream through every cell of body and brain, has a direct effect on health and behavior; that alcohol slows down and impairs ones reasoning power, memory and reflexes; that alcohol releases inhibitions, causing us to do and say things we may later regret; that before anyone takes the first drink he should be well aware of the facts, the risk and responsibilities involved, as well as the effect it might have on friends and loved ones, not to mention the pocketbook; that "drinking" is *not required* by society, nor is it necessary for a happy and successful life.

They should know how to recognize, understand and have sympathy for the alcoholic. They should dread alcoholism as they would dread polio, cancer or diabetes. Here are a few facts published by The National Council on Alcoholism:

"The social, moral and financial losses occasioned by the deterioration of 5,000,000 adults are staggering. Contrary to general belief, the great majority of the alcoholic population of 5,000,000 are not visible "skid-row" type of alcoholics found in the Monday morning court line-ups, in the jails and in the City Hospitals. Some 85% of all alcoholics are to be found in the homes, factories, offices and communities of America;

they still have families, and are still employable; often they have exceptional skills."

As we read through this report we come across some facts that should make every stockholders and industrialist vitally interested in supporting any local program for the prevention and treatment of alcoholism. It goes on to say: "Wage losses through absenteeism in industry due to excessive drinking are computed at \$432,000,000 per annum. In addition, the loss of valuable personnel, who fall victims to alcoholism after years of investment, in their training, is costing industry an astronomical amount every year."

#### TREATMENT

We are wrong when we lecture or scold the alcoholic, calling him (or her) "no good," "lazy" and "weak-willed." There is an answer now for the alcoholic who says: "What can I do about it? Where can I get help?"

Alcoholics Anonymous was founded in 1935 and from humble beginnings has grown to a sober and successful membership of over 300,000 with over 9000 separate groups functioning in all parts of the world. Its success is based on the fact that alcoholics work with alcoholics. Here there is understanding and example. Their program is based on spiritual principles and twelve important steps to a "new way of life." "Easy does it, first things first, and just stay sober one day at a time, asking God for help in the morning and thanking Him at night." In Maine there is an Alcoholics Anonymous Group in every town, city and institution of any size. AA has the highest known rate of recovery of any type of therapy in use today for the treatment of alcoholics.

Since 1949, many States have been assuming their responsibilities in this matter of education and rehabilitation. Much help, information and guidance has been provided by the *National Council on Alcoholism* and the *Yale Center on Alcohol Studies*, founded a few years previous. Public acceptance has been much slower in coming.

Under the Maine State Department of Health and Welfare, the Division of Alcoholic Rehabilitation came into being through legislation in 1953. Since that time, through research, study, trial and error, much has been learned about rehabilitating alcoholics, and edu-

cating the public about alcoholism. The Division now has five full time Counseling Centers: Portland, Augusta, Waterville, Lewiston and Brewer. At any of these counseling centers the alcoholic and/or members of his family can have their questions answered with sound advice, which if carried out (and herein lies the key) can start the alcoholic on the road to recovery. Counselors at these centers are trained and experienced in working with alcoholics and their families. Each interview is treated with strict confidence. Their methods of rehabilitation are based on diagnosis, counseling, treatment, and referrals to other specialized agencies or professions, where indicated.

*The alcoholic must first want to find sobriety.* Usually we can help him (or her) reach this decision with patience and insight, not by scolding or preaching. When the alcoholic is ready to do something about his problem he should not feel ashamed to ask for help. He can start by contacting his *family physician*, clergyman, Alcoholics Anonymous or one of the State Alcoholism Counseling Centers. Yes, the alcoholic can find help for his problem in the State of Maine.

But, alcoholism being a *total disease*, requires the *total effort* of the whole community to help the recovering alcoholic to find his proper place in society. In order to successfully do this, public attitudes must be changed. This is objectively being accomplished through factual newspaper and magazine articles, and through radio and television interviews, panels and dramas.

It is time the citizens of Maine realize that unless something is done to bring this serious public health problem out into the open, every community will suffer through increased police, judicial, health and welfare costs, not to mention the broken homes, maladjusted children, highway and industrial accidents, etc. There is no end to the suffering and expense such a situation can create if allowed to continue by an apathetic public.

*Alcoholism* has been termed a disease by medical authorities and scientists working with the problem. No one afflicted with this disease should feel ashamed, *nor should they be made to feel ashamed.* But once recognized, *there is shame in not doing something about it.*

60 Parker Street, Brewer, Maine



## Case Report — Ruptured Ectopic Pregnancy

RONALD A. BETTLE, M.D.

A ruptured ectopic pregnancy can be a formidable problem, but when this occurs in a patient who refuses necessary transfusions, it can be catastrophic.

When seen in consultation eight hours after admission, this patient was in shock with a blood pressure of 80/50, pulse 120, and a hemoglobin of 6.6 gms. with a hematocrit of 24%. Dextran had been started. Blood transfusions were refused because of religious scruples. Time had been spent in trying to get her to agree to accept transfusions and another surgeon had refused to operate unless she would accept this needed treatment.

This problem was circumvented by proposing that an auto-transfusion be carried out, to which the patient and her husband agreed. She was then taken to the Operating Room and under a combination of local anesthesia and oxygen by mask, a peritoneal tap was carried out in the line of the proposed incision. Between 800 and 900 cc. of blood was aspirated into citrate bottles, filtered and given immediately. As soon as the transfusions were running, the operation continued under local anesthesia. The affected right tube and ovary were removed. Upon completion of the procedure, the patient's B/P was 130/80, pulse had dropped to 90. This was probably due as much to clamping the bleeding vessel and arresting the flow of blood as to the transfusions.

Postoperatively the patient had a moderate amount

of distress from postoperative ileus. It had been impossible to evacuate all of the blood clots because of the limited anesthesia, and it was felt that this contributed to her distress. It was interesting to follow the Hemoglobin and Hematocrit.

Day of Surgery	7/27 Preop.	a.m. 6.6	Hg. 24	Hematocrit
	Postop.	p.m. 4.6		
	7/28	4.8	16	
	7/29	5.4	17	
	8/1	6.8	22	
	8/3	7.0	25	
	8/6	8.2	29	
Postop. visit	8/29	12.		

Treatment consisted of Intravenous protein hydrolyses postoperatively until the patient was able to take an adequate diet and simple ferrous chloride.

The patient has had no difficulty attributed to the surgery or its associated problems. Her wound healed per primum, the anemia was corrected within a month on iron therapy and her general health has remained good.

This case is reported as a possible solution in the handling of intraperitoneal hemorrhage by auto-transfusion in a patient where religious scruples rule out the replacement of blood from the blood bank.

32 Federal Street, Brunswick, Maine

## Special Article

### Is This The Negligent Physician?

THOMAS G. HARVEY, M.D.

For years the American Cancer Society, in its excellent little organ "Ca," has been publishing evidence of the harmful effects of cigarette smoking. Not too long ago the surgeon general of the United States Public Health Service issued a warning about the dangers of this habit. Many articles have been published in the medical literature on this subject, perhaps the most striking of which describes the recent work of Auerbach and his associates.

In the meantime the potent tobacco industry merrily rolls cigarettes by the billion and counts record profits from higher-than-ever sales made possible by sly, sophisticated, expensive advertising designed to make every human being of teen age or better feel that he's not "with it" unless he sucks his way through at least one pack of cigarettes a day.

A few months ago Britain's *Royal College of Physicians* published "Smoking and Health," a rather comprehensive, straight-forward summary of the evidence against cigarette smoking with respect not only to lung cancer but to other lung diseases, such as bronchitis and emphysema, and to cardiovascular and gastrointestinal diseases as well. The report urged the government to take seven steps to help curb the use of cigarettes, especially by habit-forming teenagers. Remarkably enough, within a month of the date of publication of this report, measures had been taken to implement four of these steps.

No similar practical effort has been made by the medical fraternity in the United States to acquaint millions of cigarette smokers with the risk they assume by continuing to enjoy their habit. Nor, to my know-

ledge, has any official attempt been made by the AMA or any other large representative medical organization to assess the seriousness of this problem and formulate proposals to deal with it.

That Great Britain is leading the United States in this matter should not come as a surprise to us. In spite of our higher standard of living and the big fuss we make about health and welfare the British more frequently push quietly ahead and "get the job done." It may not be widely known, for instance, that in our food processing industries the use of many adulterants and preservatives are permitted which British health authorities consider dangerous and do not allow.

Every last effort is made to raise money for, and to aid by research and treatment, the victims of multiple sclerosis, cerebral palsy, mental retardation, psychosis, epilepsy, and many other diseases about which very little is known and for which there is no cure and precious little treatment. This is as it should be, because some day new doors of hope may be opened to these unfortunate people. Even better will be the discovery of means of preventing some or all of them.

And just how much time and money in comparison is being spent to help many thousands of potential victims of crippling and killing diseases we presently know the cause of, or at least a contributory cause of? More good could be accomplished with the health dollar in the field of educating people about the poisonous effects of nicotine, arsenic, and tobacco tars absorbed in cigarette smoking than in any other field of preventive medicine I know of.

A recent letter to Food and Drug Administrator Larrick, asking why, with all the clamor about stilbestrolized chickens and sprayed cranberries, no fuss was ever made about the poisonous substances in cigarettes, brought the reply that because tobacco was neither a food nor a drug the FDA had nothing to say about it. The administration, however, does concern itself with cosmetics. It's not quite clear to some folks whether cosmetics are food or drugs.

Politicians who are willing to risk the wrath of the powerful tobacco industry are as uncommon as they are extraordinary. We cannot therefore anticipate that further amendment of the Federal Food and Drugs act of 1906 will originate with politicians. But we, as members of great medical organizations, both the AMA and the AAGP, can take the initiative in sponsoring legislation in congress to place tobacco under the watchful eye of the FDA. The very least we should do would be to solicit the support of the Department of Health and Welfare and of the health commissioner in Augusta to aid in designing and enacting legislation of this type in Maine.

To those who object that the case against cigarettes has not been proved the obvious answer, of course, is that sensible people do not wait for 100 percent proof before disclaiming a habit that has even half a chance of proving detrimental to their health, or even lethal,

later on. When the odds are even that there's typhoid in the well, do you guzzle the water until someone proves conclusively that the bugs are there? The hell you do! You get your water somewhere else, or drink beer, until the experts convince you the bugs are definitely not there.

In Great Britain only half as many doctors smoke cigarettes as do males in the general population (24 vs 50 percent). Statistics show that physicians in our own country are also becoming concerned since the number of cigarette-smoking physicians is steadily declining.

Now, even though the M.D. may not himself be convinced of the bad effects of cigarettes, he is duty bound to inform his patients that there is mounting evidence of the harmful effects of cigarette smoking. How can he in good conscience do otherwise? By pointing out to grammar school and high school boys and girls the possible harmful, perhaps lethal, end results of acquiring the tobacco habit, these youngsters will not be forced to say at some later, less fortunate, year in their lives, "We didn't know. Nobody told us."

And the older ones, those who have the habit well established (the one-pack-plus-a-day-for-years smokers), can be told that statistically, at least, their chances are better with cigars and pipes. (Can't you just see your wife gnawing at a King Edward and Aunt Cynthia forcing draft through a Dunhill? Hate to admit it but many of us can remember when it was shocking to see a lady smoke even a cigarette.)

Any talk with patients about smoking will undoubtedly be more effective if made by the physician without a cigarette in his hand. This, of course, reminds us of the most important reason why many physicians do not proscribe cigarettes to their patients — it is too embarrassing for them to condemn a habit which they subscribe to themselves.

How much more helpful it would be if President Kennedy's enthusiasm and influence could be transferred from the Medicare nonsense to this important, practical problem. It is heartening, at any rate, to know that the President has taken a step in this direction and recently appointed a committee to investigate the dangers of smoking cigarettes and make a report in a year.

One thing I am sure of — that sooner or later the majority of cigarette smokers will become concerned enough about the actual and possible consequences of their habit to do something about it.

Lung cancer will kill as many people in the United States this year as there are men, women and children in Bangor, Maine.

The ethics of our profession does not condone negligence.

Is the physician who does not warn his patients about the dangers of cigarette smoking negligent? Is this I, that no man in medicine should wish to be?

## Critique Of Certain Measures Presently Employed In Managing Patients With Cardiac Infarctions\*

"Of the various aspects of management . . . , three have been chosen as being topics of spirited controversy. They are diet, anticoagulants, and the smoking of tobacco.

"It seems reasonable to say . . . on the basis of known facts, that a radical change in the dietary habits of all persons in the United States is not now justified. Perhaps until further investigation has thrown more light on the problem, there should be moderate curtailment of intake of fats by the general population, with reasonable substitutes of poly-unsaturated for the saturated variety. Such procedure is suggested, without guarantee, as a possible means of preventing atherosclerosis or retarding its course, if already present.

"*Anticoagulants:* In the light of conflicting opinion what stand should the physician take? As in the case of diet, the lay public is aware of anticoagulant therapy, and he who refrains from prescribing is subject to blame if the course is unfavorable. Should hemorrhage or cardiac rupture occur, the responsibility likewise is his. As of now decision as to the course which the physician chooses to follow must rest with him.

"*Smoking:* In my opinion, except for persons known to react badly to nicotine, those who are dependent on smoking for curbing their emotions may be permitted to smoke in moderation; this is taken to mean ten cigarettes in twenty-four hours. The harm in excessive smoking should be stressed. The use of tobacco is strictly forbidden for at least four months after an episode of acute cardiac infarction. There should be no smoking in the presence of congestive failure. Peripheral vascular disease is an indication for permanent abstinence."

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(Levy, Robert L., American Heart Journal, Volume 64, pages 1-5, 1962.)

\*Submitted by the Maine Heart Association, Inc.



DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## 1962 Rabies Control Plan In Maine

So far as we know, we have had no native cases of rabies in Maine since a horse died of rabies in 1959 at Garland. However, we do have at least a potentially highly dangerous situation on our Maine-Quebec border. During this year there have been approximately 50 proven, or highly probable, cases of rabies in foxes and domestic animals roughly distributed along Route 24 between Daaquam and St. Gedeon, P.Q. In general, this is the area north of Coburn Gore. Three cases have been on our border, and the others have been from five to 20 miles from the border. There may be, or may have been, some unrecognized cases among wild animals on the Maine side of the border. If such is not the case, one can assume with certainty that we will get cases if effective action is not taken now. There is every reason to believe that infected foxes are the reservoir of the disease in this area, and are the animals chiefly responsible for its spread. How the disease was introduced into the fox population of southern Quebec is probably unknown. This is not the disease called "mange" which has been prevalent in our own foxes for several years. The situation is an emergency since proper action taken now may prevent introduction of the disease into Maine or may eliminate it if it already exists. Once the disease becomes firmly or widely established, elimination, or control, is an extremely expensive and prolonged effort. Introduction of rabies into Maine would have very direct, practical, and personal importance to everyone, but most particularly to sportsmen, campers, woods workers, farmers, livestock owners, and rural dwellers. Such people are most likely to encounter wild animals. The rabies virus can infect any mammal, and any animal so infected can transmit the virus via his saliva to any other mammal.

The spread of rabies can be controlled. It can be prevented in domestic animals by proper actions, and it can be prevented in exposed humans by proper treatment. Once symptoms of the disease appear, it is uniformly fatal in about 10-12 days. In general, an infected animal can transmit the disease only during the period extending from a few days before the onset of symptoms to its death, for the virus is present in his saliva only during this period. In general, too, the disease is spread only by an animal bite inflicted during the period when the biter's saliva is carrying rabies virus. One factor which complicates control is the highly variable and unpredictable length of the incubation period of the disease in animals. It may vary from

the usual 20 or 30 days to as long as six months. During such a period an animal may wander far from the point where it was infected. This also means that a bitten domestic animal not previously immunized must be carefully quarantined for a period up to six months before he can be considered to have escaped being infected. The only alternative is disposal of the animal.

Having been bitten by infected animals, pets, particularly dogs and cats and to a lesser extent other domestic animals, are the animals most likely to transmit the disease to humans because of the frequency of their association with humans.

There are five major elements in an effective control program:

- (1) Public education and information.
- (2) Reduction of the fox population to the point where spread among them is unlikely, as they constitute the reservoir of infection.
- (3) Control of stray dogs and cats. In the instance of dogs, the enforcement of dog licensing laws is important.
- (4) Immunization of pet dogs and cats, farm, and sporting dogs.
- (5) Proper handling or treatment of bitten animals or humans.

For control purposes, it can be assumed that rabies immunization of cats and dogs will give good protection for a year. However, if an immunized animal is bitten, or probably has been bitten, by a wild or unidentified animal, the veterinarian will probably recommend another dose of vaccine as a booster. In the instance of bites on other domestic animals, such as horses, cows, sheep, pigs, etc., by wild animals, or unidentified dogs, a veterinarian's advice should be sought immediately.

Our control plan is going into effect immediately and has been developed in conjunction with:

Department of Inland Fisheries and Game  
Department of Forestry  
Department of Agriculture  
Federal Fish and Wildlife Service

Control measures of some degree, depending on circumstances, will have to continue until the disease is eliminated from a reasonable area along the Quebec side of our border. Our plan is as follows:

- (1) To distribute informational material very widely.
- (2) To warn people, particularly those in the N.W. quarter of the State, that a case of rabies may be encountered.
- (3) To urge people, most particularly those in the N.W. quarter of the State, to immunize pet dogs and cats.

Note: The usual "shots" given to dogs and cats do

not protect against rabies. These are usually for distemper.

- (4) To urge, again most particularly those in the N.W. quarter, the careful enforcement of the existing dog licensing laws and the control of strays.
- (5) To urge particularly that farm and sporting dogs be properly protected by recent rabies immunization.
- (6) To alert the veterinary and medical professions to the situation.

The above will be accomplished by news releases, bulletins, pamphlets, etc. The more specific parts of our plan are:

- (1) To provide necessary diagnostic laboratory service. Diagnostic evaluation will have to be done most thoughtfully.
- (2) To maintain stocks of serum, and Semple and duck-embryo types of vaccine for free distribution for treatment of humans. Supplies of these will be in Augusta, and in the hospitals at Jackman and Farmington.
- (3) To establish a Rabies Danger Area\* roughly defined as an area six to ten townships wide along the Maine, New Hampshire and Quebec border, extending from Lac Frontier, P.Q. southward to Upton, Maine. This area will be posted with warning signs. It has been defined with the expectation that it will enclose any wild animals that may now be infected, and with the further expectation that it can be managed as a buffer strip that is wide enough so the crossing of it by an infected wild animal from Quebec will be unlikely. Use has been made of natural barriers, e.g., Moosehead Lake, the Dead and Kennebec Rivers — insofar as possible. To eliminate any animals that may be infected and to convert this into an effective buffer strip several things must be done:
  - a. No unimmunized dog should reside in this strip, or be brought into it, unless he is strictly and continuously under control and observation, such as being penned or kept on a leash. Stray dogs and cats should be eliminated. Local law enforcement and dog officers have been instructed to enforce licensing laws and the elimination of strays. To aid in this, posters to this effect will be widely distributed in the area. Free immunizations will be offered to all dogs in the area of Rockwood, Jackman, Stratton and Coburn Gore.
  - b. Extreme care should be used in handling any animal acting abnormally and any sick animals should have veterinary attention. Children should be cautioned against going to the aid of such animals. Care and judgment must be used in the case of any animal that has been, or may have been, bitten by another animal. Medical and veterinary advice should be freely sought. Posters and bulletins will be posted widely for this purpose.
  - c. Animal bites of humans, or other animals, should be reported to physicians, local health officers, game wardens, and lumber camp operators, who will have proper forms for this pur-

pose, or directly to the State Department of Health and Welfare. Each will then be evaluated and necessary advice given. Bites of humans will have to be carefully and wisely managed, and the animal doing the biting will also have to be carefully handled.

- d. To create an effective buffer strip, the fox population must be reduced and kept at a low density as long as necessary. It must be kept low enough so that there will be little likelihood an infected fox will have an opportunity to bite another fox during the 10-12 day period that he may be excreting virus in his saliva and thus pass the disease along. The quickest, most economical and most effective way to reduce the fox population is by poisoning by trained persons. Therefore, in the northern portion of the buffer strip, from Lac Frontier, P.Q. to Parmachenee Township, we plan, with the permission of landowners to reduce the fox population by a poisoning program beginning September 17, 1962. This will be conducted by five trained men from the Federal Fish and Wildlife Service, co-operating with us, plus five assistants to be employed locally. The methods to be used will be standard ones which have been successfully used in other states for years without mishap. Appropriate warning signs will be posted throughout the area. The work will be completed in two to three weeks and the method is such that no residual poisoned material will be left indefinitely in the woods. Heads of dead animals will be sent to the laboratory to help determine the extent of disease distribution, if any. It is expected that few wild animals other than foxes will be killed. No poisoning will be done in the areas about communities such as Rockwood, Jackman, Eustis. In these areas, a trapper will be employed to reduce local fox populations as much as possible. Both the local trapping, and poisoning will probably have to be repeated in the spring. A very low fox population and a solidly immunized dog and cat population are the absolute requisites for an effective buffer strip. The effectiveness of this strip as a buffer may well determine whether we do or do not have a statewide problem. Failure of this strip to protect could be disastrous.
- e. Unusual care should be taken to prevent the mechanical transportation of animals in their incubation period across the buffer strip. This means that particular care should be taken to prevent the entry into Maine of unimmunized cats and dogs that have had any possible exposure to rabies in Quebec or other endemic areas. Notification to this effect will go to customs officials.
- f. Heads from animals acting abnormally should come to the State Diagnostic Laboratory in Augusta for diagnosis.
- g. There will be continuous evaluation of new information that may become available, and the effectiveness of this plan to determine whether

\*Detailed maps of this area may be obtained by writing to: Division of Communicable Disease Control, Health and Welfare Department, State House, Augusta.

modifications of, or additions to, the program are required. Our situation should not give rise to undue alarm, or hysteria.

Our last proven case of rabies was in the fall of 1959. This single sporadic case involved a horse in the town of Garland. The last proven case prior to this involved a fox in the town of Sorrento in 1948. These two isolated cases suggest that we may have been having an extremely low rate of endemicity of the disease in the state perhaps existing since the outbreak of 1934. Other than these two cases, we have had no indication

of the disease in Maine in recent years. With such a history, we can hope that our present plan will accomplish two purposes:

(1) Eliminate any infected foxes that may exist in the buffer strip.

(2) Create conditions which may be expected to prevent the introduction of the disease into Maine from Quebec, and its consequent spread into populated areas.

If any infection has already extended into Maine from Quebec, we have no reason to believe that it has extended beyond the area chosen as a buffer strip.

PREVENTION OF RABIES IN MAN  
GUIDE FOR SPECIFIC POST-EXPOSURE TREATMENT

Nature of exposure	Biting Animal*		Recommended treatment** (in addition to local treatment)
	At time of exposure	During observation period of ten days	
I. No lesion; indirect contact	Rabid	—	None
II. Licks: (1) unabraded skin	Rabid		None
(2) abraded skin, scratches and unabraded or abraded mucosa	(a) healthy	Clinical signs of rabies or proven rabid (laboratory)	Start vaccine at first signs of rabies in the biting animal
	(b) signs suggestive of rabies	Healthy	Start vaccine immediately; stop treatment if animal is normal on fifth day after exposure
	(c) rabid, escaped, killed or unknown		Start vaccine immediately
III. Bites: (1) mild exposure	(a) healthy	Clinical signs of rabies or proven rabid (laboratory)	Start vaccine at first signs of rabies in the biting animal
	(b) signs suggestive of rabies	Healthy	Start vaccine immediately; stop treatment if animal is normal on fifth day after exposure
	(c) rabid, escaped, killed or unknown		Start vaccine immediately
	(d) wild (wolf, jackal, fox, bat, etc.)		Serum immediately, followed by a course of vaccine†
(2) severe exposure (multiple, or face, head, finger or neck bites)	(a) healthy	Clinical signs of rabies or proven rabid (laboratory)	Serum immediately; start vaccine† at first sign of rabies in the biting animal
	(b) signs suggestive of rabies	Healthy	Serum immediately; followed by vaccine; vaccine may be stopped if animal is normal on fifth day after exposure
	(c) rabid, escaped, killed or unknown		Serum immediately; followed by vaccine†
	(d) wild (wolf, jackal, fox, bat, etc.)		

\*This schedule applies equally whether or not the biting animal has been previously vaccinated.  
\*\*See explanatory notes, below.  
†Course of vaccine to be followed by supplemental doses of vaccine of non-nervous tissue if possible, 10 and 20 days after the last dose.  
(From World Health Organization, Expert Committee on Rabies, Fourth Report)

Local treatment of wounds  
All bite wounds and scratches by animals should receive immediate local treatment. For bite wounds, thorough cleansing with soap or a detergent (Zephiran or similar detergent) and flushing of the wound may be supplemented by the judicious use of concentrated nitric acid in puncture wounds where the

site permits. Where possible, bite wounds should not be immediately sutured.  
In those patients the nature of whose exposure requires the use of serum a part of the serum dose should be infiltrated into the tissue beneath the wound, when this is feasible.  
The application of ordinary antiseptics and antibiotics or

antitetanus procedures should follow the local treatment recommended above, when indicated.

*Explanatory notes to the guide for post-exposure treatment*

The general principles on which the guide is based are that in mild exposures a course of vaccine following the above-recommended local treatment is sufficient, whereas following severe exposures, and in all cases of unprovoked wild animal bites, antirabies serum together with vaccine should be employed. As with vaccine alone, it is important to start combined serum and vaccine treatment as early as possible after exposure. Serum should be administered in a single dose (not less than 40 International Units per kg of body-weight) at the start of treatment, followed by a course of not less than 14 daily doses of vaccine. In all cases where serum is followed by a full course of vaccine it is suggested that two supplemental doses of vaccine be administered at 10 and 20 days following the completion of the usual vaccine schedule. Where possible, these supplemental doses should be with a vaccine of non-nervous-tissue origin.

Sensitivity to serum should be tested before serum is used.

It is fully recognized that this table is only a guide and in

certain situations specific conditions may warrant modifications, e.g., exposure, especially in young children or where a reliable history cannot be obtained, and particularly in areas where rabies is known to be enzootic even though the animal at the time of exposure is considered to be healthy. Such cases may justify treatment immediately in a modified way. Possible modifications would be that, following local treatment of the wound as described above, a single dose of serum or three doses of vaccine at daily intervals, and no further vaccine, be given as long as the animal stays healthy for 10 days following exposure.

Another example of a local situation in which a modified interpretation of these recommendations may be indicated is that of rabies-free areas where frequent exposures to animal bites are encountered. In such localities, adequate laboratory and field experience indicating no infection in the species involved may justify the local health authorities in recommending no specific anti-rabies treatment.

(From World Health Organization, Expert Committee on Rabies, Fourth Report)

## SPECIAL COMMITTEES 1962-1963

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*(Continued from August issue)*

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\*Appointed by the Council of the M.M.A. on 6/19/62

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1. Best, C. H., and Taylor, N. B.: *The Physiological Basis of Medical Practice*, ed. 6, Baltimore, The Williams & Wilkins Company, 1955, p. 578.

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# County Society News

## KNOX

September 4, 1962

The Knox County Medical Society held its first meeting of the fall season on September 4, 1962 at the Knights of Columbus building in Rockland, Maine. The social hour and dinner at 6:30 p.m. were followed by the business meeting. The main subject of discussion was community group inoculation by oral polio vaccine, which program is supported by this society.

Harold N. Willard, M.D., Director of Chronic Care and Rehabilitation at Thayer Hospital in Waterville, was guest speaker for the evening. Dr. Willard presented interesting and informative ideas on the important community and hospital problem of "Rehabilitation."

MUSTAFA V. ONAT, M.D.  
*Secretary*

## LINCOLN-SAGADAHOC

September 18, 1962

The regular meeting of the Lincoln-Sagadahoc County Medical Society was held at the Ledges in Wiscasset, Maine on September 18, 1962. There were fourteen members and one guest present.

Charles E. Burden, M.D. and Alexander G. Stetkevych, M.D. both of Bath were elected to membership in the society. Paul A. Fichtner, M.D. of Bath was elected to membership by transfer from Franklin County Society.

Robert H. Eddy, M.D. of Rockland was the guest speaker of the evening. Dr. Eddy's subject was "Rehabilitation of Strokes."

GEORGE W. BOSTWICK, M.D.  
*Secretary*

## CUMBERLAND

September 20, 1962

Seventy-two members and guests were present at the Cumberland County Medical Society meeting which was held at the Eastland Motor Hotel in Portland, Maine on September 20, 1962. The meeting was called to order by the President, Robinson L. Bidwell, M.D.

Robert A. Bearor, M.D. and Norman W. Saunders, M.D. both of Bath were elected to membership in the society and Richard I. Clark, M.D. of Freeport by transfer from Lincoln-Sagadahoc County Society. Elected to service affiliate membership was Captain David Naide.

The obituary of DeForest Weeks, M.D. was read and it was voted that these be spread on the records of the society and a copy sent to Mrs. Weeks.

A lengthy report by the Committee to Review the Size, Make-Up and Mode of Operation of the Council of the Maine Medical Association was read by Robinson L. Bidwell, M.D., Committee Chairman, and discussed by our councilor Thomas A. Martin, M.D. A motion that the delegates be instructed to vote in favor of the measures embodied in this report was tabled until the next meeting and it was voted that in the interim a copy of the report be sent to each member of the society.

Boris Vanadzin, M.D. discussed the proposed delay in the polio clinics until spring. President Reznik of the Junior Chamber of Commerce stated that the Chamber felt that there

was no reason for delaying the clinics in view of the statements of Dr. Dean Fisher and the United States Health Service approving the use of type 3 vaccine. After much discussion, participated in by many members of the society, it was the consensus that the clinics be delayed until the spring and Dr. Vanadzin was authorized to give a statement to the press following consultation with the officers of the society.

Elton R. Blaisdell, M.D. announced that Diabetes Detection Week would be held at the Maine Medical Center this year and asked support of the Cumberland County Medical Society which was approved.

Dr. Bidwell urged the members to attend the Fall Clinical Session of the Maine Medical Association which will be held at the Eastland Motor Hotel in Portland, Maine on October 26, 1962 and sponsored by the Cumberland County Medical Society.

ALBERT ARANSON, M.D.  
*Secretary*

## KENNEBEC

September 20, 1962

The Kennebec County Medical Association held its monthly meeting at the Augusta House in Augusta, Maine on September 20, 1962 with the President, Loring W. Pratt, M.D., presiding.

The Report of Committee to Review the Size, Make-Up and Mode of Operation of the Council of the Maine Medical Association, Robinson L. Bidwell, M.D., Portland, Chairman, was presented by Paul H. Pfeiffer, M.D. of Waterville and commented upon by Ernest W. Stein, M.D. of Pittsfield. Samuel H. Kagan, M.D. of Augusta asked that the resolution favoring the fluoridation of public water supply passed by the Kennebec County Medical Association in May be reconsidered. The motion was defeated by all but one vote.

Samson Fisher, M.D. of Waterville presented colored illustrations of various dermatologic conditions of interest to everyone.

EARLE M. DAVIS, M.D.  
*Secretary*

## PISCATAQUIS

September 20, 1962

A meeting of the Piscataquis County Medical Society was held at Dr. Linus J. Stitham's Sebec Lake Cottage on September 20, 1962. All members were present except Albert M. Carde, M.D. who was recovering from an operation at the New England Baptist Hospital in Boston. Guests present were: Drs. Allan Woodcock, Thomas H. Palmer, Jr., Wilbur B. Manter and Donald Coulton all of Bangor and Paul R. Briggs, M.D. of Hartland.

Dr. Stitham moved that the Piscataquis County Medical Society go on record not to sponsor the Sabin Oral Vaccine at this time — each individual doctor to make his own decision as to the use of the vaccine. This motion was passed unanimously after being seconded by Charles H. Lightbody, M.D. of Guilford.

A resolution sponsored by Ralph C. Stuart, M.D. of Guilford, President of the Maine Medical Association and seconded by Dr. Stitham was passed. This resolution to be sent by

the secretary to the Council of the Maine Medical Association:

"The Piscataquis County Medical Society wishes to go on record as being entirely in agreement with the Kerr-Mills Bill and its principles. We concur as a body that we are in disagreement in the way the bill is administered in the State of Maine under the control of one individual."

Dr. Lightbody informed the society of the Blue Cross Plan to sell insurance to those over 65 years and that Maine will not be included as the plan was turned down by the Blue Cross of Maine.

The society passed a resolution instructing Dr. Stitham to vote against the plan pertaining to the Report of Committee to Review the Size, Make-up and Mode of Operation of the Council of the Maine Medical Association, Robinson L. Bidwell, M.D., Portland, Chairman.

The following slate of officers were elected for 1963:

President, Francis W. Bradbury, M.D., Dover-Foxcroft

Vice-President, Linus J. Stitham, M.D., Dover-Foxcroft

Secretary-Treasurer, Isaac Nelson, M.D., Greenville

Delegate to the Maine Medical Association House of

Delegates: Linus J. Stitham, M.D., Dover-Foxcroft. Alternate: Charles H. Lightbody, M.D., Guilford

Board of Censors: George C. Howard, M.D., Guilford

(3 yrs.); Norman H. Nickerson, M.D., Greenville

(2 yrs.) and Ralph C. Stuart, M.D., Guilford (1 yr.)

Legislative Committee: Harvey C. Bundy, M.D., Milo (3

yrs.); John B. Curtis, M.D., Milo (2 yrs.) and Nor-

man H. Nickerson, M.D. (1 yr.)

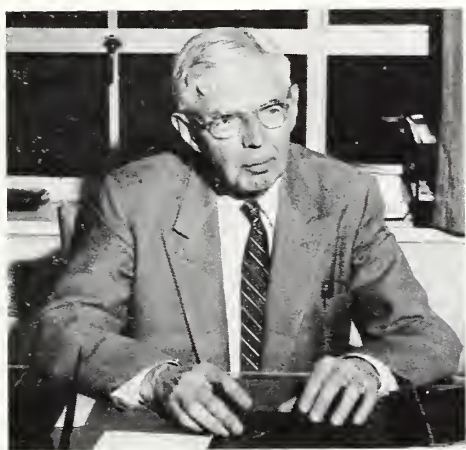
ISAAC NELSON, M.D.

Secretary

## News, Notes and Announcements

### Maine Physician In The National Scene

Frederick T. Hill, M.D., medical director at Thayer Hospital in Waterville, received the highest commendation — Honorary Fellowship — from the American College of Hospital Administrators, a professional society with headquarters in Chicago.



DR. HILL

The tribute to Dr. Hill took place at the College's 28th Convocation ceremony in the Arie Crown Theater at McCormick Place before more than 2500 members and guests. The presentation was made by Tol Terrell, president of the ACHA and administrator of the Shannon West Texas Memorial Hospital in San Angelo.

Dr. Hill was one of five persons to be honored by the college for their contributions to the health and hospital fields here and abroad.

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Ramon Angeles, Jr., M.D., D.C. General Hospital, Washington, D.C.

Abbas Bashirelahi, M.D., 746 Colorado Avenue, Louisville, Kentucky

Carlos Benjamin Fernandez, M.D., 438-52 Street, Brooklyn, New York

Margarete J. Hoch, M.D., 83 So. Fullerton Avenue, Montclair, New Jersey

Hubert Jockin, M.D., 44 Brookstreet, Brookline, Massachusetts

Yutaka Kikkawa, M.D., Bronx Municipal Hospital Center, New York 61, New York

Gerard Lapointe, M.D., St. Leonard, New Brunswick, Canada

Sanghwan Lew, M.D., 380 Pearl Street, Burlington, Vermont

Denis Mazerolle, M.D., St. Leonard, New Brunswick, Canada

Eric Finlay Nicholas, M.D., 30 Pepperell Street, Halifax, Nova Scotia

Alex Niedzwiecki, M.D., 7 Carroll Street, Portland, Maine

Takashi Okagaki, M.D., 245 Pond Avenue, Brookline 46, Massachusetts

Herman Polet, M.D., Peter Bent Brigham Hospital, Boston 15, Massachusetts

Roberto Quero, M.D., 10390 Main Street, Clarence, New York

Hans Rasch, M.D., 716 North Street, Pittsfield, Massachusetts

Walter Rohm, M.D., 105 Pleasant Street, Concord, New Hampshire

Javier Sagarminaga, M.D., Montreal Neurological Institute, Montreal, Canada

Pedro Antonio de Oliveira e Silva, M.D., Robert Packer Hospital, Sayre, Pennsylvania

Andrew Christopher Walsh, M.D., 6200 Riverdale Avenue, Riverdale 71, New York

### THROUGH RECIPROCITY

Edwin H. Abrahamsen, M.D., 74 Malin Road, Malvern, Pennsylvania

Martin A. Barron, Jr., M.D., Portland, Maine

Gerald E. Callery, M.D., 324 Wayne Avenue, Lansdowne, Pennsylvania

James E. Cavanagh, Jr., M.D., 388 State Street, Portsmouth, New Hampshire

Frank S. Cruickshank, Jr., M.D., 141 Maple Street, Needham, Massachusetts

J. Warren Harthorne, M.D., 10 Forest Road, Cape Elizabeth, Maine

Alfred Hurwitz, M.D., 2404 Avenue K, Brooklyn, New York

Hisashi Majima, M.D., c/o K. Sugino, 136 Hicks, Brooklyn, New York

Thornton W. Merriam, Jr., M.D., 114 Shelburne Road, Burlington, Vermont  
 Eugene P. Montgomery, M.D., 1648 8th Avenue, Greeley, Colorado  
 Robert Milton Morrison, M.D., 40 Berkeley Street, Portland, Maine  
 Anders T. Nerland, M.D., 317 State Street, Bangor, Maine  
 Warren Dean Pope, M.D., 3104 Market Street, Wilmington, Delaware  
 Mohamed S. Saydjari, M.D., 132 W. Division, Barron, Wisconsin  
 George Truchly, M.D., 408 First Avenue, New York 10, New York  
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# The Journal of the Maine Medical Association

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No. 11

## The Recognition And Management Of Depression\*

WILFRED DORFMAN, M.D.\*\*

With 19,000 suicides yearly, failure to recognize a depression may be as disastrous as failure to diagnose an acute coronary occlusion. Many depressed patients are seen by non-psychiatrists rather than by psychiatrists. In some instances this is due to a shortage in supply. In others it is better related to the key position held by the family physician and to the fact that somatic symptoms and/or illness obviously lead the patient to seek medical help at the usual sources.

The recognition of depression may be simple and obvious at a glance, yet at times it may be difficult and easily missed. The patient may deny it because of conscious or unconscious motives. He may be ashamed, feeling that his symptoms are a sign of "weakness"; he may thus prefer an organic diagnosis rather than face this supposed stigma. If he happens to have suspicious or proven organic illness his depression will find a most acceptable outlet in this direction, not only for himself but unfortunately too often for his physician.

### TYPES OF DEPRESSION

There are many types of depression; their recognition will make for better management and treatment.

Reactive or neurotic depression is a state to which man is universally vulnerable. Tragedy will strike and losses will occur to which the human organism must

react. Acute grief reactions require time to repair the trauma that is produced. If reactions persist beyond a reasonable period of time, it is possible that an endogenous depression, coming from within the person, has been superimposed. The differential diagnosis of neurotic and psychotic depression may thus be difficult at times.

Manic-depressive illness is a cyclic affair. The premorbid personality is cyclothymic, with its characteristic shifts in affect from periods of elation to periods of depression. As the aging process proceeds, depressions may become more prolonged; precipitating events may become blurred and indistinct. There is a characteristic early awakening, a loss of weight and a danger of suicide. Milder cases are not necessarily psychotic.

Involuntional depression is characterized by agitation, depression and many somatic complaints. It occurs during the involuntional period; there is no previous history of depression. The premorbid personality is frequently obsessive-compulsive.

Senile depression may be related to both cerebral arterio-sclerosis and the normal frustrations that accompany the realization that lifelong ambitions and plans may not be accomplished.

Schizoaffective disorders combine the features of schizophrenia with those of depression. Diagnosis here may also be difficult and delayed unless the schizophrenic process is readily evident. Apathy and depression often defy differentiation.

Depressive equivalents often include somatic symptoms, with or without somatic disease, which frequently mask a depression. The clue here often lies in

\*Presented at the 109th annual meeting of the Maine Medical Association, Rockland, Maine, June 18, 1962.

\*\*Assistant Attending Physician, Department of Medicine; Assistant Attending Psychiatrist, Maimonides Hospital of Brooklyn, New York, and Clinical Instructor, Department of Psychiatry, Downstate Medical Center, New York.

the failure of both specific and non-specific remedies to alleviate the somatic difficulties and should serve to alert the physician to the possibility of a masked or hidden depression, despite the fact that the patient may deny it vehemently.

#### DEPRESSION IN ORGANIC ILLNESS

Depression may precede roentgenographic or other evidence of carcinoma of the pancreas or lung; it is on occasion found as a symptom of brain tumor, cerebral arteriosclerosis or general paresis. In lupus erythematosus, depression has been related to organic brain changes. In rheumatoid arthritis, obesity, duodenal ulcer, ulcerative colitis, and some dermatological disease, depression may at times alternate with the somatic illness. Rapid cures may thus disturb psychic homeostatic mechanisms sufficiently to precipitate a psychosis. Pregnancy and various endocrine states are often associated with depression. Addison's disease and Cushing's syndrome, despite the fact that they represent opposite poles endocrinologically, are capable of producing depression. The same is true for hypopituitarism and acromegaly, hypo and hyperparathyroidism. Hypothyroidism can both mimic and produce a depression.

In somatopsychic depression, the diagnosis of a somatic disease is the trigger that sets off a reactive depression. Coronary disease and malignancy both carry tangible and realistic threats to one's existence, and may precipitate a depression. Trauma and viral disease, especially if accompanied by long periods of inactivity may be threatening to patients with a strong need for activity and can similarly set the stage for reactive depression.

Iatrogenic depression can follow a surgical procedure if the patient is insufficiently prepared emotionally. A hysterectomy, herniotomy, plastic surgery, mitral commissurotomy, etc., all carry the risk of emotional sequelae.

Depression can follow the use of drugs such as reserpine, ACTH and steroids. In the case of reserpine, it has been related both to the biochemical effects of a lowering of serotonin and norepinephrine as well as to the psychodynamic effects of tranquilization in a personality where this produces a threat because of the loss of activity. ACTH and steroids can produce emotional sequelae due to a lowering of the serum or red cell potassium level and are correctible by electrolyte balance. There are also psychodynamic effects. Here the disruption of psychic homeostasis, where sudden cure may conflict with the secondary gains of illness, plays a major role. Clinically it accounts for the disturbing emotional upheavals seen after successful steroid treatment of rheumatoid arthritis and skin disease, where crippling disease had become a way of life.

A clinical evaluation of depression should probe its depth, length and differential diagnosis. A family or personal history of depression may offer clues to the possibility of manic-depressive illness. The depth, in-

dicated by severe sleep disturbances, loss of appetite and loss of weight will often suggest psychotic rather than neurotic difficulties. The presence of severe agitation, when coupled with somatic complaints and severe depression should suggest an involutional psychosis rather than an anxiety state. Early awakening, diurnal variations (worse in morning, better at night) may also point to a psychotic process.

Generalized somatic complaints, especially when they are clearly disproportionate to the organic findings, may indicate the presence of a depression. These complaints are usually vague and poorly localized. Frequently they follow no known disease pattern. Attempts to explore a symptom in depth is often blocked by vagueness, denial and poor verbalization. Difficulty in communication and lack of accessibility may aid in pointing to a psychotic rather than neurotic depression.

Symptoms of autonomic imbalance are frequent. Dryness of the mouth, headache, hyperhidrosis, tachycardiac and visceral spasms are frequent. Fatigue is a predominant symptom and is most significant when it occurs soon after awakening and improves towards the end of the day. Difficulties with dentures is a frequent symptom.

Facial expression, posture and lack of gestures all present clues to the alert observer. In some instances, however, these clues can be masked by a disarming smile.

Loss of interest, motivation and concentration are seen in all types of depression; severe loss may indicate a psychotic state. Helplessness and hopelessness are similarly greatest in the more severe psychotic depressions. Expressions of guilt, excessive self-criticism and evidence of retroflected rage and expiatory behavior are seen in all depressions, the degree varying with the severity. Anxiety may cover up a depression and emerge as it lifts. In some instances patients are treated with tranquilizers which may increase the depression.

Loss of sexual drive, slowed thinking, a loss of ability to verbalize and a lowered self esteem are seen in all types of depression. Here too the depth of the depression can be gauged by the severity of these symptoms.

Clinical evaluation includes an assessment of possible psychodynamic factors. The feeling of loss may be real or symbolic. Death, separation due to illness, financial losses or loss of health are realistic and potent precipitating agents. Equally potent and realistic, though less apparent, is sudden success with its attendant fear of envy and loss of love of others. Anniversary reactions to a parent's death may mobilize self-destructive feelings due to unconscious guilt feelings.

Introjection and identification refer to the Freudian concept of depression. The needed love object or person is "introjected" and "incorporated"; unfortunately feelings for the needed person are ambivalent, (a mixture of love and hate) representing both the basic need and the necessity to deny it.

Hostility and feelings of aggression are repressed and relegated to the unconscious. Since they cannot be expressed, their mounting energy seeks its only available outlet and is retroflected against oneself. Self-punishment, and the need for expiation are the inevitable result.

Many depressed patients show obsessive features; a peculiar omnipotence of thought produces guilt feelings as if thoughts are lethal. Resentment towards a parent or parental figure, a natural sequel to dependency and need for placation, becomes translated into a "death wish" when the parent dies.

Oral eroticism refers to the oral dependency needs of depressed persons. Their needs for approval, attention and love are often excessive and have been compared with the needs of an infant sucking at its mother's breast. These narcissistic needs are often insatiable, so that minor disappointments produce severe reactions — as if the loss were a major event. Self esteem is therefore easily disturbed since it is almost totally dependent upon outside sources for its nourishment and replenishment. It is difficult indeed for mates, employers or friends to cater to these infantile needs — especially when attempts to do so are rejected by the patient.

#### THE TREATMENT OF DEPRESSION

Psychotherapy has different meanings to many physicians; this is related to basic differences in orientation, philosophy, attitude and goals. It must include a meaningful patient-doctor relationship which eventually results in the patient's increased ability to adapt to life and its problems. In the management of depression, supportive and reparative techniques have been shown to be more efficacious and less damaging than those attempting a radical dissection of the personality. Reparative psychotherapy should have as its goal the alleviation of the acute situation through ventilation, reassurance, encouragement and support. Fear, anger and guilt will then recede in many instances, and bring with it a return to emotional homeostasis. Reconstructive techniques should be left to the trained psychiatrist and psychoanalyst. They are contraindicated even here during the acute stages of depression, but may be necessary in the management of chronic neurotic depression. Flexibility in technique permits the well trained therapist to respond to the patient's needs rather than to use a fixed plan or approach. There are times when the doctor must listen, permitting himself only an occasional

grunt, at other times a more active participation is essential, in order to establish communication and meaningful contact.

Electroshock therapy is still the treatment of choice in severe depressions, especially where suicidal ideation is present. It is rarely indicated in neurotic depression, unless an endogenous root is suspected. Manic-depressive states, involutional depressions, schizoaffective and senile depressions all respond to electroshock. In many instances 4-6 treatments will suffice. The advantage of electroshock over drug therapy lies in its speed and reliability; drug therapy may often take 4-6 weeks for a full effect and is not as predictable as electroshock. Drug therapy has many potentialities, but it also has its pitfalls. In neurotic depression, amphetamine derivatives and methylphenidate are still superior to many of the newer psychopharmaceuticals. The monoamine oxidase inhibitors may be of help in retarded depressions. Imipramine has its greatest value in endogenous depression while amitriptyline is clearly of great value in agitated depressions. Although some of these newer drugs seem to have biochemical effects and probably correct faulty enzymatic or metabolic dysfunction, these possibilities still await further clarification. It is nevertheless fairly certain that many of the newer anti-depressants have curtailed the need for electroshock in many instances and have lessened the number of treatments when used concomitantly. It is also fairly well established that anti-depressants can be synergistic with psychotherapy; they often enhance communication and increase the ability of the physician to reach the patient.

Most important is the question of psychiatric referral. The era of the new drugs has provided the non-psychiatrist with additional means to help manage depressed patients. He must nevertheless remain aware of the fact that drugs alone do not suffice. With an increased understanding and knowledge of the psychodynamics of depression he should be better equipped to handle many of these patients; yet in some instances, especially when suicidal ideation is present, or he feels that he cannot effectively reach the patient, psychiatric opinion and/or referral may be a necessity.

An understanding of basic psychodynamics is essential, but it must be coupled with the usual and expected attributes of the good physician: the desire to help and the awareness of individual limitations both in himself and his patient.

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1921 Newkirk Avenue, Brooklyn 26, New York

# The Conquest Of Inner Space\*

BERTHA PHILLIPS RODGER, MD.

A fantastic amount of newspaper space has been allotted to the attempts to conquer outer space. An inconceivable number of dollars have been spent on preparations, for making rockets, training astronauts and all the rest. Much time, thought, fear and worry are centered upon it. A definite amount of progress is visible. We can point to it with pride, compare it with that of the neighbors. The accomplishments to date are truly marvelous. The potentialities are as yet unfulfilled. It is vivid, dramatic. It captures the imagination, lifts us above the humdrum of daily existence, turns us toward new horizons.

Except for paying taxes, watching it on TV and cheering from the sidelines, most of our own personal lives are more intimately woven with another conquest, that of inner space. Since this touches us daily in a constant struggle, it is a battle frequently without glamor, that goes on with little support and a discouraging lack of appreciation of its meaning and its potential. We find ourselves plodding along in leaden boots, shoulders bowed by the weight of unresolved conflicts, contradictory expectations, and confusing demands from within and from without. Anxious, sick, exhausted by the struggle, wondering what it all means — if anything, — the patient turns to the doctor for a magic pill to cure his ills. How he longs to orbit, weightless, above the earth, freed from the limitations imposed by his human existence!

The doctor himself longs for some magic that will free his patient from this dreary and painful setup. He reads eagerly of each new drug or therapy, searching hopefully. He is tempted to proffer a pill with the hope that it will have the desired magic effect. He is faced with a strong temptation to play God, for this is a further role foisted upon him by the public's image of him. Indeed, his own person may get lost in the shuffle. He may get to believe himself that "Doctor knows best." He finds that his busy practice demands every shortcut. He may then use the authoratative approach even to the extent of encroaching upon the rights of the individual to make his own choices, choosing for him, thereby increasing his dependency.

One of the therapies dredged up from the past and refurbished for the present is Hypnosis. Not a therapy in itself, it is rather a means by which therapy can be applied, a vehicle or a tool. It is sometimes presented as a two-edged tool but this is a false assumption. If it seems to be two-edged, it is not so by its own nature,

but rather by virtue of the user. Hypnosis is an interpersonal relationship. As such, it has two possibilities. It can be exploiting, demanding subservience, undermining self-confidence, increasing a feeling of unworthiness, used as a bludgeon, or perpetrating indignity to the person. It can be used in an opposite way, as an integrating force, in a manner that is healing and unifying, increasing independence and the assumption of responsibility for the self, teaching self-discipline. The former use is a magic one. It is dramatic, sensational. It is a kind of playing God, trying to dominate. It is then exhibitionistic and may even be sadistic. As such, it is a danger. This is unscientific, un-medical use. It arouses great anxiety. Medical use is quite different. It offers no magic, but something far more constructive and lasting.

*Homeostasis:-* Despite the accumulated scientific studies on homeostasis, we still do not know how this takes place. Yet the body itself knows, better than any physician can describe, the best way in which to stabilize and repair itself. Putting it at rest without the necessity of drugs which may interfere with respiration or circulation presents a distinct advantage. This seems to be especially true when it is accompanied by a quietness of mind wherein the patient feels comfortable and at ease. To some extent, physical and emotional ease may go hand in hand. Both may be enhanced by suggestion. Even in the Hypnoidal State the patient may be told,

"You breathe all the way out . . . and rest . . . your lungs fill spontaneously with good clean air . . . the oxygen in it is carried to every part of your body, every cell . . . wherever it is needed. Waste products are picked up and brought back to be breathed out with every breath. Thus your body goes on renewing itself constantly. This is a built-in ability. It goes on without your having to direct it. It continues even while you are asleep at night . . . or under an anesthetic. It changes automatically with every changing need. You can alter it voluntarily when you want to but it's so wonderful to know you need pay no attention to it. You can trust it, just enjoy its soothing, easy rhythm, relaxing deeper with each breath until you are very very comfortable. It goes all through you. You give yourself up to that lovely, easy feeling. The less you try, the better you succeed. That brief rest period for the muscles at the end of each breath is enough to refresh them so they can keep right on breathing for you. Your body knows how to heal itself. Re-creation goes on all the time. Resting and relaxing helps it to do so. Stay relaxed until your tray is brought in . . . and wake up feeling wonderful!"

\*Presented at the 109th annual meeting of the Maine Medical Association, Rockland, Maine, June 18, 1962.

Restlessness may use up energy and become a drain on the body's resources. Most people respond to illness and discomfort with restiveness which usually includes uneasiness of mind. Teaching repose of body is reflected in increased calmness. It may come as a surprise to a patient to discover that he can learn to apply this art himself with great benefit. So often he remarks, "This is something I've needed to do for a long time but I never knew how!"

*Heeding Minimal Warnings:-* Learning to pay attention to the early minimal warnings given by the body may minimize disability which follows failure to heed them.

A man of 48 had recurring attacks of pain in the lumbo-sacral region. These were severe enough to keep him in bed for five days at a time. Sometimes he would be aware that his back was "going to go out," as he expressed it, but he could do nothing to avoid its development. Often the attacks would come without warning when he stooped to pick a nail off the floor. X-Ray showed a congenital defect of a lumbar vertebra. The orthopedic surgeon recommended a brace and discussed spinal fusion. The patient refused to consider either, electing to see what hypnosis could offer.

The pain-spasm cycle was given as a rationale. Whatever the cause of pain, the response is likely to be a protective spasm of the muscles in the involved area. This is a natural response of a reflex sort. The sudden tightening of muscles can be very uncomfortable and even hurt. The added pain induces more spasm. Thus a vicious circle is set up. Anything which can break the cycle gives it a chance to calm down. Learning to respond by relaxing lessens the spasm, greatly increases the comfort. Simple relaxation alone can change the response to pain as much as 40%. Paying attention to the first warning signals avoids aggravation of the area.

These things were explained to the patient in a light trance. Posthypnotic suggestion was given that

"Stored away in your mind are memories of all the attacks of pain you have had. Your conscious mind may not remember which movements of your body brought on these attacks but this knowledge is filed away in the subconscious. You can review in your mind even the memories of the physical aspects, how the beginning felt, what movements made it worse what positions. You can learn to pay attention to these first small warnings. You can respond by stopping motion at once in an unfavorable direction. When spasm occurs, you can allow it to relax as soon as your position is properly adjusted. You can review your memories and understandings in such a way that you can utilize them at once and put them into operation whenever you need to in the future."

Although he continues to complain from time to time that his back "feels as if it might slip," he has lost no time from work. There have been occasional times when he was willing to lie flat in bed and read for a few hours to give his back a needed rest. There have

been no restrictions necessary on physical activities. He has learned to pay attention to the early warnings given by his body and to respond in a constructive way, becoming more independent as a result, freed from the limitations his body had imposed upon him.

*The Patient's Own Accomplishment:-* A woman was seen for relief of persistent urinary retention following a vaginal repair for stress incontinence. For sixteen days she had been unable to void despite exhortations, warm water poured over the perineum, the sound of running water, encouragement to void in the shower, hot sitz baths, and all the tricks of the trade.

A light trance was induced. She was asked to

"Remember when you were a little girl . . . some pleasant occasion when you were so busy you just didn't want to stop and go to the bathroom. Remember the feelings — but don't act on them yet — when you suddenly realized you'd have to find a toilet fast . . . an increasing desire to void, a tingling. Soon you'll feel a sense of mounting pressure within . . . and all the other feelings. You will go into the bathroom settle down comfortably . . . and then you may act on those sensations . . . experiencing a feeling of relief. You will have whatever tensions you need, just the excess ones will be relaxed. Your bladder knows exactly how to empty itself. It will do so very efficiently and comfortably. I can tell you how to do it, I cannot do it for you, but you can do it and you will feel wonderful afterward."

Later she told the nurse, "I felt so sleepy while Dr. Rodger was talking to me, but she didn't do a thing for me. All I could think of was how much I needed to go to the bathroom. I just wished she would leave so I could go." and she did — after sixteen days of inability to do so!

This patient accepted suggestion and took over for herself. She quite rightly gave the credit to herself. By her own changed attitude, she had become independent. We were both content with the result.

*Replacement of a Childish Reaction with an Adult One:-* A young woman, admitted with profuse bleeding following an incomplete abortion, was first seen in the Operating Room where she was to have a D + C. Cyclopropane was started with a hypnotic technique of softly orienting the sensations to be expected into a setting of the familiar and the pleasant. Induction was rapid and smooth. A very small quantity of agent was needed, recovery was easy. She was fully reacted within fifteen minutes of removal of the mask. After a brief period of observation in Recovery Room, she was returned to her own room.

The next day she reported, "I didn't think I could ever take gas again. When I was only four I had a terrible experience with it. A dentist gave it to me without ever telling me what was going to happen. He and the nurse just held me tightly in the chair. He clamped a big mask tightly over my face and held it there while I cried and struggled and wet the chair in my fright.

Oh he was angry! He was really mean to me afterward. My father was away then. It was during the war. My mother couldn't do anything about it. I felt so all alone and so imposed upon. I trembled whenever I saw a dentist for years after and wouldn't go near one.

"When I saw you waiting there with the mask in your hand I thought I'd better not say no to you then, so I went ahead. It was easier than I thought. You made it seem easy, talking to me like that, explaining it. Now I'm glad to find out I really could go through with it."

"You feel it's important that you were able to lay that ghost? You feel strengthened to discover you can choose the way you respond. You don't have to act like a frightened child every time."

"Yes, that's it. I don't think I'll ever have trouble like that again. I seem to have found out something about myself."

*Amnesia an Aid to Quiescence:-* A woman in labor with her third baby was reacting out of proportion to the mildness of her contractions. It was deemed too early to give medication, lest contractions be stopped. Her last baby was Rh negative and died two days after birth. The mother was very apprehensive. She was glad to have someone talk soothingly to her and readily went into a trance.

"You can use your imagination to help you be more comfortable. Picture some safe place where you can lock things up. You can get at them any time you really need them. Nod your head when you see it. Perhaps it would be all right for you to take any memories of previous pain and store them away carefully. . . You might also like to put away any painful thoughts . . . and painful expectations. Look around to be sure there's nothing else you want to put away. When everything is stored away, close it up, lock it, and put the key where you can easily find it when you really need it. You can appreciate that this labor and delivery are different for any other one. You can look forward to a normal one and something very nice to show for your efforts. You have time to re-live any pleasant memories you would enjoy. . . You may have any medication you need as you go along . . . as you give yourself up to relaxing with it, you will be much more comfortable. . . Let the time pass quickly and pleasantly."

Her response was excellent. Her labor and delivery proceeded without complications. She was delighted that she had been able to carry through with a minimum of difficulty. The replacement of her emotional turbulence with quietude by the constructive use of her imagination was a maturing experience which she greatly appreciated.

*Minimizing discomfort:-* A boy of twelve was to have an emergency appendectomy. The premedication given to bring him to the Operating Room in a calm frame of mind had not succeeded in abating his apprehension about needles. The 20cc syringe of Pentothal looked alarming to him. The nurse's coaxing to "take it like a man" only aroused further uncomfortable feel-

ings. He was becoming more and more wide awake and closer to the edge of panic.

He was told, "I would like you to keep your eyes firmly fixed on that syringe over there on the table. Do not take your eyes off it for a second. Make sure no one touches it. Would you be willing to help me by counting seconds for me? Begin with twenty and count down very slowly. Now repeat, a little more slowly."

Meanwhile, an intravenous was started in his opposite arm, without his being aware of it. Posthypnotic suggestion was given for breathing deeply and relaxing during the operation, for rapid recovery afterward and for complete comfort. Pentothal was injected as this was finished.

The boy was pleased to be thanked the next day for cooperating so well. He was delighted that it had been so much easier than he had anticipated.

Many diagnostic and therapeutic procedures cause great apprehension to the unknowing, especially to those who have accepted all the wrong suggestions from those in their environment. Just a little re-direction can alleviate this distress. Not the least of the benefits is the patient's own participation and the strengthening effect that conquering apprehension has.

*Dissociation:-* One of the simplest and quickest ways to minimize an uncomfortable procedure is to teach the patient how to utilize dissociation. This is something most children and many imaginative persons do spontaneously. Some even use it in the wrong way. Guided, it can be learned or directed into constructive channels to yield a more comfortable response.

A very apprehensive multipara had bulging membranes. This seemed to be all that was holding up progress. The obstetrician wanted to do a sterile vaginal exam and rupture membranes. The patient, however, cried out each time she was touched and complained bitterly of rectal exams, tensing up so that little information was obtained. Using Dr. Coulton's technique, she was shown how to dissociate to the beach where she could enjoy a sunbath. Relaxation was excellent. Membranes were ruptured with no disturbance to the patient. Afterward the patient said, "I'm so glad you helped me to help myself. I was so ashamed before. I knew I was acting like a baby but I didn't know what to do about it. I don't want to be like that!"

*Counterbalance:-* While some people regress to childish behavior, others hold themselves under such tight rein that they are afraid to relax at all lest the results be devastating. It is a great relief to them to learn how to develop a counterbalance so that they will not go to extremes.

A young executive complained of multiple pains, the distribution of which was anatomically impossible. He was afraid of medication which might interfere with his alertness on the job. He was referred for hypnosis as an alternative to tranquilizers.

He had chest pain, palpitation, tachycardia, and a feeling of "farawayness" at times. All of these symp-

toms were distressing to him. Being of an analytic turn of mind, it was not easy for him to learn to respond to suggestion. With hand levitation, pendulum counting, and other mechanical types of technique, he did enter a light trance, surprised to find he really could relax safely. He was willing to practice at home and to review his daily program to see what might need re-arranging.

At the next visit, he discussed the recent airplane trips he had been taking frequently over the past many months. These bridged time changes which were upsetting to him. Biological rhythms were discussed and the need for making allowances for changes and getting "in tune" with them instead of expecting the body to adjust automatically just because clock time was altered.

The complaint that his mind was too busy when he tried to relax was countered in two ways. It was also suggested that he might concentrate on feeling a pleasant warmth in the solar plexus which radiated and spread to all of the body in a soft glow, leaving the forehead cool. He was reminded of the Prayer for Quiet Confidence in the Book of Common Prayer,

"Oh God of peace, who hast taught us that in returning and rest we shall be saved, in quietness and in confidence shall be our strength; By the might of thy Spirit lift us, we pray thee, to thy presence, where we may be still and know that thou art God . . . Amen."

He went much deeper into trance at this time. He was delighted to find he could bring this about himself and could awaken himself after a set interval which he could vary to suit his needs.

His next report was that he was doing very well. The previous discomfort seldom bothered him. He was no longer afraid to relax, feeling a security in being able to control the time and depth enough to feel very comfortable about it. The feeling of refreshment after such a rest seemed incredible to him. In over five months, it has not been necessary for him to see his family doctor except for a minor laceration. "I feel sort of like a musical instrument that has been tuned up, tightening some of the loose strings and loosening others so the tones are again true. I like to feel I am controlling myself, not giving in to outside forces nor dependent on drugs. Before I learned to relax I was strung too tightly. It was a miserable feeling."

*Intellectual Quietness:-* Mental quietness can be taught as well as physical. To some extent, the two go hand in hand for it is always in his entirety that man reacts, that he is either tense or relaxed. Quietness may be attained by setting the stage for it physically, narrowing down awareness, centering down from outward distractions to inner concentration, or any way suited to the particular need of the moment and the ability to respond at a particular time.

Our understanding of the mind is limited. Various analogies have been used to help form a working concept. Each one is limited. The simile of the iceberg

with the major part submerged below the level of consciousness is vivid but tells only a small portion of the facts. The mind has been likened to a storage warehouse, where experience and understanding is filed away. Perhaps a more dynamic concept is that of a river into which a torrent pours, which has various currents constantly bringing material to the surface. Debris seems to get washed up haphazardly and in unrecognizable form. Deep within, there seems to be a sort of radar-like scanning device, according to Cheek<sub>1</sub>, which every living organism has. This tells it what to avoid, what to go toward, what to use for food, what to eliminate. The response comes forth after the stimulus has been processed by a sorting system that far outranks the F.B.I. finger-print-matching system. It is cross-matched with all experience and understanding, according to some inner evaluation. Deepest of all, there seems to be a well spring of inner life, this core of values, inner faith and hope, a creative spirit, a divine spark.

This deep storehouse of inner treasures can be called upon in time of stress. The subconscious learnings are a part of them. We need to learn more about how to call them into play. We need to learn to trust intuition, these unconscious learnings, how to use them intelligently. We do know that the deliberate induction of quietness can set free intellectual as well as physical resources. Before a mechanical computer can be set for a new problem, it must be cleared of a previous one. It is as if a moment of stillness does this for the unconscious mind. The reminder that every bit of understanding and experience the person has ever had can be used now, somehow seems to free intellectual forces. It frees them from less important matters that are tangential and interfere. Letting the mind have a period of stillness is rather like setting milk aside in a cool place, the cream rises to the top. Nearly everyone has had the experience of going to bed at night with an unsolved problem which leaves him feeling frustrated. He has awakened in the morning with the solution. The subconscious mind knows better than the conscious mind how to make the necessary connections and to work through a problem even during physiological sleep. This ability can be utilized in intellectual matters, in problem-solving, decision-making, and the like. It is enhanced by direction and suggestion from another. It can also occur in response to auto-suggestion.

A resident was to take the written examination which constituted the first part of her specialty exams. She had been out of medical school for several years, having taken leave until her children were grown enough to be in school. Although her preparation had been conscientious, she was distressed at having so much dependent on these exams. In trance she was reminded, "You have done the required work and reviewed it thoroughly. You have excellent training and clinical experience. All the information acquired is stored safely in your subconscious mind, ready to be coordinated and

brought forth in response to the questions as they are presented. You will read the questions carefully, then relax deeply. You have plenty of time in a few seconds of clock time to review all of your knowledge and understanding pertaining to each question and to arrange it for clear presentation. Your mind works with the speed of thought. When you put pen to paper, the pen will write for you. Your thoughts will flow freely. Enjoy allowing this to happen."

She was the first of the group to be finished with the exam. She read it over carefully, surprised to find how much more neatly and legibly it was written than was usual for her. Finding no fault with it, she turned in the book and went out. Her feeling was that of relief that it was accomplished so quickly and easily — and wonderment that it seemed to go so well. Marks were not given, only the report of passing or failing. A year later, however, one of the examiners informed her, "You did exceptionally well on your written exam. I remember it well though I did not know you at the time!"

The mind is more versatile, more flexible, more dependable than any machine ever invented. We ought to learn to trust it, to develop and use its capacities.

*Spiritual Quietness:-* There is still another kind of quietness that is even more vital. It is the kind of quietness described in the Prayer for Quiet Confidence quoted before. In a world vibrating constantly with insistent clamor, distractiveness, and discord, there is a desperate need for calmness, composure, and tranquillity. Yet we seek them too often in the wrong places. They are not so easy of attainment as the "happy pill" ads would lead us to believe. There is a way of calling forth a sense of deep stillness that lets healing, integrative forces flow freely.

A woman of 60 was hospitalized for cardiac evaluation following an attack of chest pain. She was troubled about many things, her physical symptoms, family problems, and emotional difficulty. She was sleeping poorly and feeling worried without being able to feel satisfied as to the cause of the worry. She had sought help and felt that things were no longer out of control. "Oh if I could only be really quiet for a while I think I might feel better! There's so much hustle and bustle and my mind is so over-busy that I just can't seem to settle down."

It was difficult to find a way for her to relax. Her attention was frequently distracted. She kept analyzing every move instead of giving herself up to responding. Finally, hand levitation was accomplished and brought forth that she really liked that feeling of lightness. She liked the idea of floating or walking along the beach in a filmy dress in a gentle breeze. It suddenly became easy to capture her imagination and put it to work. She did

go into a nice trance, fairly deep. She was reminded of the Prayer of Quiet Confidence and that she could retire at any time into "the little chapel of the heart" as St. Theresa puts it, even in the presence of others for the quiet she so much needed. She would have plenty of time in the hospital to consider in detail the meaning for her of the Twenty-Third Psalm, "The Lord *is* my Shepherd . . . He planned for me long ago . . . planned that my body can repair itself . . . that it can respond in time of trial . . . He maketh me to lie down in green pastures . . . where it so quiet and so peaceful and calm . . . I can remember even brief moments of such calmness and live them over again . . . the remembrance of past joys and happiness . . . the anticipation of future ones . . . the memory of loved ones . . . of far horizons that give perspective . . . these He has planned to help tide me over difficult times . . . I need fear no evil in His strength. . . ."

A few days later she reported, "I had forgotten what deep peacefulness is available to those willing to respond. I've been so busy about unimportant things that I've let the important ones slide. Now I see what it is I must do and I know I will be able to do it."

There is a peace that passes all understanding. Before the doctor can mediate it to another, he must have experience of it himself. There is no technique nor magic formula for it. It comes by personal encounter. It is something which is caught rather than taught. It is a channel through which flow the healing and integrating forces which lead to the true conquest of inner space.

#### SUMMARY

Here, then, are four aspects of the conquest of inner space which can readily be put to daily use. All have to do with the induction of quietness and may have overlapping and far reaching beneficial by-products.

1. Physical quiet brings repose and alertness to body signals.

2. Emotional quietude replaces turbulence and is strengthening and maturing.

3. Intellectual quiet can clear the mind and aid in the intelligent use of intuition, utilizing the mind's capacity more fully.

4. Spiritual quiescence brings rest and confidence, strength and deep peacefulness.

May the peace that passes understanding be yours to enjoy sharing.

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# The General Practitioner And The Alcoholic Patient

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The problem of alcoholism may seem alien to many physicians in the practice of general medicine. Alcoholism is an immensely complicated illness in which psychological and social factors sometimes overshadow the medical component. Its management is admittedly difficult. The alcoholic in the acute phase is thought to require the facilities and special staff of a hospital, and the long-term treatment of the chronic alcoholic is the responsibility of psychiatrists, social workers, specialists connected with private and public sanatoria, clinics, rehabilitation centers and hospitals devoted to this group of patients, Alcoholics Anonymous, and churches.

Because of changes in our concept of this disease, and recent gains in the chemotherapeutic approach to the problem, the general practitioner will undoubtedly play an increasing role in treating alcoholics. Before we discuss the medical aspects of these changes, we must outline the magnitude of the problem. There are at least five million known alcoholics in this country, and a large proportion of them are receiving little or no effective help. Alcoholics Anonymous is doing a magnificent job with its 250,000 members — but this is only 5% of the total. There are not enough psychiatrists and other specialists to supply the need for prolonged treatment of our growing army of uncontrolled drinkers. If they are to receive help, we can no longer delegate their treatment entirely to the specialists, and must call the family doctor into the emergency.

This is a move in the right direction. The general practitioner is often the ideal person to treat the alcoholic. As a rule, he has to know the patient for some years and is familiar with his clinical history; he is known and trusted by the patient's family, and in a position to influence and counsel them in their treatment of the patient — one of the crucial factors in his therapy. Successful treatment of the chronic alcoholic is usually based on a long, close relationship with a medical man who treats him with patience and understanding. Sporadic encounters with a series of strangers on the staff of the various hospitals or "rest homes" that the alcoholic may visit, probably do him as much harm as good, because of his limited exposure to the staff. He wrongly suspects them of indifference, and this further exaggerates his feelings of being rejected and misunderstood. Hence he adds these people to the long

list of hostile authority figures that he may defy in order to protect his already weak and wounded ego.

While the physician in general practice is thus well qualified in the personal sense to treat the alcoholic, until recently there have been vexatious technical problems in the management of the alcoholic during the acute phase. Hospitalization has been desirable for this period, and will remain necessary in the event of delirium tremens or acute hallucinosis. Also severe medical complications such as endocrine disturbances, resulting in electrolyte and metabolic imbalances, pneumonia, gastritis, peptic ulcer, G.I. bleeding, liver damage and traumatic head and body injuries require hospitalization and appropriate therapy.

However, in the absence of such extreme symptoms, it is now entirely safe for the family physician to treat the alcoholic in the intoxication and hangover phase in his own home. This revolutionary statement was made in late May of 1961 by the Alcoholism Division, Department of Mental Health of the State of Connecticut, which has been a pioneer in the scientific study of alcoholism. It occurs in a letter addressed to all the physicians of the state, recommending the use of chemotherapy for the home management of the patient in withdrawal.

A sovereign state, after intensive research by its alcoholism experts, has taken a major step toward enlisting the family physician in the campaign of caring for and possibly rehabilitating the patient who has been unable to control his drinking. This is a giant step.

If the physician in general practice is to deal with alcoholism, he must attempt to understand this medico-social-psychologic complex. During the last few years the medical aspect of the complex has been receiving long overdue attention; the social and psychiatric components have, in the opinion of many, been overemphasized. Alcoholism is a disease. E. M. Jellinek, of the Yale School of Alcoholic Studies, a great authority on this ailment, in his book, "The Disease Concept of Alcoholism," offers a useful classification of the alcoholic into five main types, which delineate certain traits of clinical importance. These are:

1. *Alpha alcoholism*, an habitual, purely psychological dependence on alcohol to relieve physical or emotional distress, but with no loss of control, or inability to stop drinking.

2. *Beta alcoholism*, in which medical complications result from excessive drinking, but without physiological dependence.

\*Presented at the fall assembly of the Maine Chapter of the A.A.G.P. at the Lafayette Hotel, Portland, Maine, October 7, 1961.

3. *Gamma alcoholism*, the type most frequently found in this country. Its chief indications are withdrawal symptoms and loss of control over drinking. This type does more damage than the others to the individual's health, personal relationships and ability to earn a living.

4. *Delta alcoholism* also involves withdrawal symptoms, but the patient has not lost control in the sense that he can regulate his intake though he is compelled to drink regularly. This is the type commonly seen in the wine-drinking countries of Europe.

5. *Epsilon alcoholism*, the class to which the periodic spree-drinker belongs.

In this classification the diagnostic fulcrum is *control*. The gamma alcoholic is literally unable to stop drinking until he is too ill or inebriated or dead broke to continue. At this point one may ask: is alcoholism an addiction? One authority points out that it is entirely similar to narcotic addiction in developing withdrawal symptoms and physiological and psychological dependence, but quite dissimilar in respect to tolerance. Since the liver cannot metabolize more than 10 to 15 ml. of absolute alcohol in the course of an hour, tolerance cannot develop. Thus alcoholism appears to be a disease entity of a certain type of addiction, and Jellinek suggests that a study of the pharmacology of addiction would advance our understanding of this disease entity.

Research teams are finding various clues as to the bodily reaction to alcohol which may ultimately help to explain why certain individuals become alcoholics. E. C. Hoff suggests the possibility of genetic individual differences in enzyme activity which may affect alcohol metabolism and cause certain persons to fall into the alcoholic pattern. Hormonal imbalance is another hypothetical factor in the etiology of alcoholism, the theory being that chronically tense persons such as alcoholics do not synthesize enough adrenochrome, or destroy it too quickly, and that alcohol as a tension-dissolving agent may be used as a substitute for the missing adrenochrome. There is experimental evidence behind the various facets of this hypothesis. Studies in both animal and human subjects by Hoff and various co-workers are clarifying the effect of alcohol upon higher cerebral autonomic control mechanisms. The development of a neurotropic drug that can protect the autonomic system from higher cerebral over-response to stresses may prove to be of clinical value.

#### RECOGNITION OF THE ALCOHOLIC

While many physicians sincerely believe that there are no alcoholics in their practice, this may be a comfortable illusion. The alcoholic who has not reached the point of surrender is extremely skillful in protecting himself from detection. The physician must outwit him by watching for tell-tale signs such as the following during history-taking and examination:

Recurrent attacks of upper respiratory tract or upper gastrointestinal tract complaints without somatic indications or the presence of bacterial or viral invasion.

Superficial clues such as heavy brown furring of the posterior aspect of the tongue, a bloated appearance of the face and neck, or an odor of stale alcohol about the patient (which often leads him to avoid getting too close to the physician).

Neurological complaints such as peripheral neuritis, insomnia and tremors; and psychological signs such as over-dependence, hostility, suspicion, rebellion, and sometimes bizarre behavior indicating disturbed emotional patterns. The occurrence of the above symptoms should alert one to the possibility of alcoholism as an underlying cause. The physician of today should definitely consider the disease of alcoholism in differential diagnosis, and eliminate it before arriving at a conclusion on the patient's illness.

#### THE PROBLEM OF DROPOUT

With the best will in the world, the physician cannot treat a patient who isn't there. A major problem in the therapy of the alcoholic is the difficulty of getting him into the office in the first place, and the even greater difficulty of getting him to come back. This is true even when the patient has from childhood been willing to visit this same doctor for the swabbing of a sore throat or the splinting of a fractured arm. These are definite, socially acceptable troubles, but addiction to alcohol still bears the stigma of the days when it was considered a sin rather than a disease. This feeling has been so strong in past history, that only 50 years ago a Bath, Maine physician was ostracized from his medical society because "he suggested that the alcoholic was suffering from an illness rather than from weakness of moral and spiritual fibre." Not only is the alcoholic ashamed of his addiction, but he is in a more or less chronic state of confusion and hopelessness which makes it next to impossible for him to take the simple step of getting help. The third reason is the most powerful one: the awareness that medical intervention means the end of his drinking. The basic law so widely publicized by AA is recognized by patient and physician alike: the true (gamma) alcoholic is lost if he takes one drink, because it is literally impossible for him to stop after that first drink. The old permissive attitude of advising the patient to cut down on alcohol intake has long since proved pernicious; any doctor will tell him he must cut alcohol out altogether.

Thus the alcoholic knows that if he becomes a steady patient of any medical man his drinking is doomed. Since he is by definition unable to stop drinking, and since the one thing that matters to him is getting the next drink, it is a wonder that he visits a doctor at all. Actually, he is often forced into this action by desperate family or friends. But he manages to slide out of subsequent visits in a large percentage of cases.

While this would appear to be an impasse, there are ways of handling the problem. The physician must clear his mind of the old censorious attitude toward the chronic alcoholic, and treat him as he would any other

sick person — but a sick person in special need of understanding and reassurance. Often there are medical complications needing immediate attention, and always there are the vitamin deficiencies and other direct effects of alcoholism to be corrected. Since the patient accepts these strictly medical offices as giving him relief, and since the wise doctor does not remind him too vigorously that they are closely related to his chief ailment, a good therapeutic relationship can be built up as the basis for a later attack on the primary trouble.

SPECIAL PRECAUTIONS IN THE THERAPY OF THE ALCOHOLIC

The uncontrolled drinker does represent a clinical risk unless the physician avoids certain pitfalls peculiar to this type of patient. A few may be mentioned.

Don't prescribe large enough amounts of a drug to harm or kill the patient who is suicidal or too befuddled to know what he is doing and takes the whole amount at once. Alcoholics have a high drug tolerance, and must receive heavier than the usual dosages. They are also notorious overdosers, and should never have too much leeway in self-medication. If possible, potent drugs should be administered by a reliable member of the family. Don't make prescriptions refillable.

Never prescribe an alcoholic patient paraldehyde, amphetamine, morphine or any of the barbiturates. These are capable of producing addiction, and the alcoholic has proved to be more vulnerable to drug addiction than other types of patients.

In administering anesthetic for surgery, remember the alcoholic's drug tolerance, especially when gas or sodium amytal is used.

Don't interview a woman alcoholic, especially in the home, without a third person present.

Don't take an alcoholic's statements at face value; he will say anything to avoid detection of his addiction and protect his liberty to go on drinking. But don't confront him with his misstatements, or show fear or hostility. He has an infallible instinct for sensing reactions, and rejection by the doctor, even if carefully dissimulated, ruins the relationship.

THE USE OF PSYCHOTROPIC AGENTS

I believe that we have now mentioned the chief negative aspects of therapy in the alcoholic patient. As the general physician knows from his experience with the newer tranquilizers in many areas of medicine, they are an almost unmixed blessing. The rauwolfias, meprobamate, the phenothiazines and other classes of drugs have all been tried out in the treatment of alcoholism, especially of the acute phase of intoxication and withdrawal, and some of them have proved useful.

In my own practice with alcoholics, promazine in intramuscular injections of 150 mg. every four hours controlled the symptoms of acute alcoholism, but entailed occasional hypotension, psychomotor agitation and the recurrence of withdrawal symptoms upon discontin-

uance of the drug. Injections of hydroxyzine pamoate (Vistaril) 50 to 100 mg. every four hours proved excellent for the control of arrhythmias but not for other symptoms of the acute phase. Then chlorpromazine became the sedative of choice until the advent of chlordiazepoxide (Librium), which is revolutionizing the treatment of alcoholism. This agent has supplied the answer to many of the problems which previous tranquilizers failed to solve.

Recently I used chlordiazepoxide in a series of 75 alcoholic patients with an age range of 25 to 75 years, and the medical and psychoneurotic complaints to be found in any group of this sort. After a stat dose of 100 mg. by intramuscular injection or by mouth the patients were maintained on oral dosages of 50 mg. every three or four hours for the next 12 to 24 hours. The second day the oral dosage was 25 mg. q.i.d., and the patients were then maintained on 5 to 10 mg. t.i.d. or q.i.d. Duration of therapy ranged from 3 days to 7-and-a-half months. Side effects were minimal; drowsiness in 2 patients, drowsiness and ataxia in 2; one patient developed psychomotor agitation, and another a temporary senile psychosis with severe ataxia. In only one case did the side effects interfere with the patient's functioning, and in all but one case they disappeared when dosage was reduced.

Marked to moderate improvement was obtained in 69 of the group, or 92%. The 11 patients with acute alcoholism were all controlled, and 47 of the 51 patients with uncomplicated chronic alcoholism showed marked to moderate gains.

The effect of chlordiazepoxide on the alcoholic patient in the acute stage is to make him quiet and manageable. Usually he falls asleep, but may easily be roused. When he awakens after one to three hours he

TABLE I					
RESULTS OF CHLORDIAZEPOXIDE THERAPY IN 75 ALCOHOLIC PATIENTS					
Diagnosis	No. of Patients	Improvement		Unchanged or Worse	
		Marked	Moderate	Minimal	
Chronic alcoholism	51	26	21	3	1
Acute alcoholism	11	6	5		
Chronic alcoholism and psychoneurosis	2	1	1		
Chronic alcoholism and psychotic disorder	2		1		1
Chronic alcoholism and acute angioneurotic edema	1		1		
Acute alcoholism and psychoneurosis	3	2		1	
Acute alcoholism and gastritis	1		1		
Hysterical blindness, compli. alcoholism	1	1			
Alcoholism and epilepsy	1	1			
Unspecified	2	2			
TOTAL	75	39	30	4	2
		( 92% )			

is ready to take nourishment, his sensorium is clear and he is cooperative. Supportive treatment includes restoring electrolyte balance through the use of glucose and adrenal cortical extract, correcting vitamin deficiency by administering B<sub>12</sub> and B complex parenterally, and bed rest as long as needed.

A maintenance dose adjusted to the individual is of great value after the acute phase is over. It alleviates the post-alcoholic tension state, which usually leads to more drinking, and in a surprising proportion of cases it makes the patient amenable to measures aimed at his rehabilitation and possible permanent cure. These include individual or group psychotherapy, the services of AA and local clergy, and steady attendance at a clinic or doctor's office for sustained medical therapies.

During May of 1961 a symposium on alcoholism was held at the Colorado Academy of General Practice in Denver. Many of the unique effects of chlordiazepoxide on the alcoholic patient were reported by specialists, and some of them should be mentioned here.

First the great problem of drop-out is greatly diminished by medication with this agent. In one study using four different classes of tranquilizers matched with a placebo, about 60% of the patients on three of the agents failed to return for a second week of therapy. The fourth drug, chlordiazepoxide, was used in a double-blind study of 214 outpatient alcoholics, of whom half received a placebo. Fifty-two per cent of the patients on the drug returned the second week, as against 35% on placebo. By the sixth week 20% of the patients on drug were still coming in for treatment against 11% of the placebo group.

The physician making this study remarked of chlordiazepoxide, "It has been the most effective drug as regards return rate of any we have studied to date. This was true with both 'skid row' and 'white collar' populations, and whether or not they were 'dry' or suffering from recent drinking." He could not explain this effect of the drug except by its general action of giving the patient a sense of well-being and freedom from tension, and this influence was especially clear in those patients who had suffered relatively more than the others in feeling anxiety and fear at the start of treatment.

Another specialist, director of a state alcoholic clinic, said that after chlordiazepoxide therapy was instituted for all patients in January, 1960, patient interviews increased from the pre-medication rate of 161 a month to 437 a month by April, 1961. He added that many more patients remained in long-range continuous treatment than had done so under prior chemotherapies.

Because of the addictive tendencies of alcoholics and of many drugs habitually prescribed for them, it was interesting to hear from another expert that 46 of 80 alcoholic patients had voluntarily reduced their chlordiazepoxide dosage because they felt so well. He remarked that this reaction was especially important because alcoholics have a tendency to increase their dosage of sedative-type medication, and many of them become

addicted to paraldehyde, the barbiturates, chloral hydrate or the bromides.

Another point was that alcoholic patients reached optimum response levels on this agent much sooner than controls. At examination 40 hours after medication, 54% of the patients on chlordiazepoxide had reached this level, while only one of those on other drugs had done so. At 80 hours, 90% of the patients on the drug had reached the optimum, against 72% of the controls receiving other standard tranquilizers.

An important contribution to the symposium was made by Doctors J. E. Rosenfeld and D. H. Bizzoco, respectively psychiatrist-in-charge and senior physician of the Blue Hills Hospital, which is the in-patient center of the Alcoholism Division of the Connecticut State Department of Mental Health. For some years the medical staff of the hospital conducted studies, most of them double-blind, on both the standard and newly introduced drugs recommended for alcoholism therapy, the last of the series being chlordiazepoxide. The results seemed to them so remarkable that this drug was adopted for medication of over 1,000 alcoholics in all stages of the disease. This in turn led to the unprecedented action of a state board of health recommending a specific drug for a given purpose. The letter to the physicians of Connecticut, signed by Rosenfeld and Bizzoco, specifies that chlordiazepoxide be used for the acute phase of alcoholism, except in patients over 65 years of age, who may become confused and ataxic with the large dosage necessary to control intoxication and hangover. This dosage schedule is the one I followed in the study just reported. In stating that this drug may safely be used in the general hospital or in the patient's home by the general physician, the authors are careful to explain many of the points to which I have briefly alluded: the necessity of hospitalization for the patient in delirium tremens or acute hallucinosis, who may require some restraint and somewhat higher dosages; supportive therapy and diet; and side effects. Both the letter and the paper presented by Rosenfeld and Bizzoco mention a side effect which may be unfamiliar to physicians who have already used lower dosages of this drug in other indications: an incidence of about 5% of ankle edema. While this reaction has not proved serious, and can be controlled by reducing dosages or administration of Diuril, it suggests a disturbance in water metabolism or retention worth study.

This valuable letter of advice ends with the admonition with which all discussions of alcoholism must close: this is a curable disease, but the cure is a long-range and often arduous process requiring the skill and patience of the attending physician. When we consider the wreckage caused by the uncured alcoholic to himself, his family and society, the long hard road to abstinence is the only one we as physicians can recommend toward a goal of the utmost value to us all.

*The continued achievement of high standards of patient care in the preventive, curative, and restorative aspects of illness depends upon a harmonious, collaborative relationship between medicine and nursing. In an effort to protect and foster an enduring alliance of understanding and cooperation between these 2 major health professions, the Committee on Nursing has instituted a continuing program of liaison, communication, education, and research. The Committee has authorized publication of the following report on its objectives and program.*

VERONICA L. CONLEY, PH.D., *Secretary*

## Objectives and Program of the AMA Committee on Nursing\*

The program of the AMA Committee on Nursing is based on 3 general assumptions: (1) that nurses have a separate and distinct professional status and their contributions are those of co-workers; (2) that nursing should expect the medical profession to support and endorse high standards of nursing education and service; and (3) that each of the various levels of academic and technical accomplishment in nursing makes its own unique contribution to the total health care of the public.

On the basis of these broad assumptions, the Committee has adopted the following objectives:

1. *To expand and strengthen liaison activities between organizations representing the medical and nursing professions at the national, state, and local levels.*

Liaison has been established with all the major nursing organizations (including the American Nurses' Association, the National League for Nursing, the National Federation of Licensed Practical Nurses, the National Association for Practical Nurse Education and Service, and others) as well as with constituent and component medical associations, medical specialty groups, and several national organizations with a collateral interest in nursing.

The Committee feels that one of its major contributions is to promote interprofessional conferences between physicians and nurses. A committee composed of AMA and ANA representatives is now planning a conference on nurse-physician aspects of professional practice. The Committee on Nursing will also encourage the inclusion of nurses on programs of national and state medical meetings and attempt to remedy the scarcity of positively oriented, unbiased material on nursing in the medical literature.

2. *To study and report to the medical profession on current practices and trends in nursing and on developments among nursing auxiliary personnel.*

Through its headquarters staff, the Committee is collecting information on nursing matters vital to physicians. A file of abstracts, excerpts, and reprints is available for quick reference.

3. *To stimulate, initiate, and, where feasible, support research in areas pertinent to the nurse-physician relationship in professional practice.*

Such research requires the collaboration of many disciplines. Several nurse-physician teams are now engaged in extensive research projects. These include studies of inter-disciplinary

participation in planning care; the nursing needs of chronically ill ambulatory patients; and the amount and type of nursing service which makes the maximum contribution to maternal and infant welfare.

4. *To offer advisory services to both professions on inter-professional matters.*

The secretary and chairman of the Committee serve at present on the committee on careers of the National League for Nursing. The secretary is also a member of the advisory council of the National Federation of Licensed Practical Nurses, the National League for Nursing's committee to study costs of nursing education, and the hospital advisory council of the National Association for Practical Nurse Education and Service. The Committee will also serve as a consultant group to committees, councils, and departments within the AMA. Similar services have been offered to constituent and component medical associations.

5. *To provide support and assistance to the nursing profession and its nonprofessional auxiliary personnel in their efforts to maintain high standards.*

Nursing, like medicine, is faced with pressing demands for change if high standards are to be maintained in our present environment of rapid scientific and social advances. Nursing is now engaged in a continuous reevaluation of its educational system, its scope of services, its legal responsibilities, and other phases of its practice which reflect in the quality of patient care. This Committee supports the efforts of the nursing profession in maintaining high standards and offers its cooperation and assistance.

6. *To encourage physicians to accept invitations to serve on nursing school faculties.*

In view of growing pressures on the professional nurse to assume responsibilities of a medical nature, the teaching role of the physician warrants reevaluation. At the present time, some nursing schools are finding it necessary to assign nurse faculty members to lecture on medical subjects.

If the medical and nursing professions are to make the fullest use of their joint potential, they must have not only a common denominator of interest in the patient and a comparable body of knowledge, but also the kind of relationship that derives from a deeper appreciation of, and respect for, each other as allies working toward the same goals.

CLARENCE H. BENAGE, M.D. CHARLES L. LEEDHAM, M.D.  
ELIAS S. FAISON, M.D. WILLIAM R. WILLARD, M.D.  
BENSON W. HARER, M.D. ARTHUR A. KIRCHNER, M.D.,  
*Chairman*

\*Reprinted From The Journal of The American Medical Association. August 4, 1962, Vol. 181, Page 430, Copyright 1962, by American Medical Association.



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### The Rapid Laboratory Diagnosis Of Streptococcal Disease

CHARLES H. OKEY, PH.D.\*

As part of the campaign against rheumatic heart disease and its antecedent, rheumatic fever, the importance of early and accurate diagnosis in cases of streptococcal disease caused by the Group A beta hemolytic streptococci has been emphasized in the years since World War II. The corollary of prompt and sustained penicillin therapy has been stressed by authorities and organizations in the field of rheumatic heart disease control. Appropriate action based on these two principles has led to a reduction in the incidence of rheumatic fever and sequential rheumatic heart disease. The Diagnostic Laboratory is prepared to assist the physicians of the state in diagnosis by providing a prompt and accurate means of establishing whether patients whose symptoms are suspicious of streptococcal disease do actually have an infection with Group A streptococci.

The investigations of the Army Epidemiological Board during World War II established the relationship between Group A streptococci and rheumatic fever. While the mechanism whereby streptococcal disease in some patients may lead to rheumatic fever remains obscure, prompt and accurate penicillin therapy prevents the mechanism from operating. A similar preventive influence is obtained against the occurrence of acute glomerulonephritis following streptococcal disease.

The accurate diagnosis of Group A streptococcosis from among the many types of upper respiratory tract infection is generally considered to be difficult if not impossible without laboratory assistance. Laboratory studies indicate that among the cases with the broad designation of upper respiratory infection, only 10% to 20% have been shown to be due to the Group A streptococci. Sharpening the criteria to include only exudative pharyngitis and/or tonsillitis, about 30% can be demonstrated to be caused by Group A. The remainder of the cases are caused by various viral and bacterial agents. Some of these bacteria are those strep-

tococci belonging to groups of the beta hemolytic kind other than Group A. These organisms are capable of producing severe illness but rheumatic fever is not observed as a later complication. It is the laboratory function to isolate and identify quickly and accurately the causative agent in cases of suspected streptococcal illness. The time factor is important in that the general recommendation by various authorities suggest that appropriate therapy must be started within the first 10 days of illness in order to prevent rheumatic fever. The accuracy of the identification of the agent gains importance from the observation that as many as 40% of the blood agar plates showing beta hemolytic colonies are not actually Group A streptococci and belong to other streptococcal groups. Prolonged penicillin therapy for patients suffering from infection with these latter organisms would be unnecessary and entail some expense and a risk of drug sensitization.

For many years the designation of beta hemolytic streptococcus as Group A was laborious, time-consuming and expensive. Occasionally, the laboratory report was available too late for use in a particular case. A major break-through in laboratory identification became available two years ago when the fluorescent antibody technic was demonstrated to be suitable for streptococcus identification. Through the cooperative efforts of the Communicable Disease Center and the Heart Disease Control Program groups of the United States Public Health Service, state and local personnel were trained in the technic and funds provided to place appropriate equipment and reagents in each of the state health department laboratories throughout the country. The Diagnostic Laboratory has been providing this service since last January on a small scale and is now ready to make the test available as requested on a wider basis.

Specimens for throat culture should be taken by using the KL or swab outfit and firmly passing the cotton swab over a diseased area. These should be mailed without delay to the laboratory but if it is anticipated

\*Director, Diagnostic Laboratory.

that the specimen would arrive on Sunday they should be refrigerated and then forwarded for Monday delivery. Specimens received on a given day will be reported the following day by mail. Physicians wishing to receive reports earlier than by mail should so indicate on the laboratory slip and authorize a collect telephone call for the report. Procedures in the laboratory are the preparation of a pour-streak blood agar plate from a broth in which the swab has been placed, incubation overnight and observation for beta hemolytic colonies typical of streptococci. The swabs are refrigerated until the following day. Pour plates are advantageous in detecting the occasional culture that appears weakly hemolytic or non-hemolytic when surface streaked. The swabs corresponding to plates showing typical colonies are then incubated for four hours, the swab discarded, the broth centrifuged and the sediment suspended in saline. Smears are made on special slides, the Group A specific antiserum coupled with fluorescein is added and incubated for 30 minutes. Examination of the specimens together with appropriate controls is carried out with a microscope using an ultra violet light for illumination. Streptococcus of Group A fluoresce brightly and constitute a positive finding; streptococci of other groups either do not fluoresce at all or fluoresce weakly.

Despite the fact that rheumatic fever is a preventable disease, cases continue to occur here in New England and in Maine. Czoniczer, Lees and Massell<sup>1</sup> reviewed the medical histories of 105 patients with acute rheumatic fever recently admitted to the House of the Good Samaritan in Boston. The review was designed to determine the principal symptoms and thus to make recommendations for diagnosis and therapy.

None of the patients had received adequate penicillin therapy at the time of the antecedent streptococcal infection. Therapy was not given in 69 cases because the patients had not been seen by a physician. This was understandable in 16 cases in which the illness was entirely subclinical but in 38 cases the illness was mildly symptomatic and in 15 cases the disease was fairly severe. Of the remaining 36 patients, 14 did not receive

appropriate therapy because of incorrect diagnoses and 22 either were not given penicillin at all or it was given in inadequate amounts.

Of the 89 patients in whom the disease was clinically evident the chief symptoms and their frequency were as follows: sore throat and fever, 58; fever without sore throat, 17; sore throat without fever, 4; respiratory symptoms without sore throat or fever, 10. Fever with sore throat was recorded in 84% of the 89 patients with symptoms; sore throat without fever occurred in only 4 cases.

The authors conclude their discussion with the following recommendations:

"Whenever a child does not seem well in any way, his mother should take his temperature four times daily.

"If definite fever (temperature of 101° F. or more by mouth) is present, a physician should be consulted.

"Unless the cause of the fever is obvious the physician should have a throat culture taken. If this culture is found to be strongly positive for beta hemolytic streptococci, it is likely that the illness is due to a streptococcal infection.

"When definite fever is accompanied by a strongly positive culture, penicillin should be administered in adequate dosage. Such dosage can be provided by a single injection of 1,200,000 units of benzathine penicillin or by 400,000 units of orally administered penicillin three times daily for ten days."

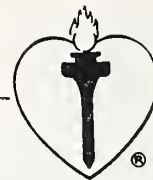
In using the Diagnostic Laboratory service for streptococcus isolation and identification please bear in mind that:

It is not necessary to examine all patients in an outbreak or during periods of high prevalence because:

1. The general area or group involved will show one predominant type; and
2. Close associates of a case (e.g. household contacts) will be found infected with the same type whether or not they exhibit a clinical syndrome. Judicious selection of a few patients with the same general clinical picture will be found, most often, to have the same strep type; and this finding can be rationally extended as etiologic definition for a much larger number of patients in the same locale.

<sup>1</sup>Czoniczer, G., Lees, M., and Massell, B.F.: Streptococcal Infection. The New England Journal of Medicine, Volume 265, Number 19, p. 951-952, November 9, 1961.

## Maine Heart Association Notes



### Recent Advances In The Treatment Of Arrhythmias And Conduction Defects

#### Digitalis toxicity

"Because of the high mortality rate in some types of digitalis-induced arrhythmias and the new advances in the treatment of digitalis toxicity, it is of the utmost importance that this diagnosis always be entertained by the clinician. The problem of intoxication has been increased by the popular use of purified glycosides of digitalis and increased reliance on oral saluretic drugs. Diuretics cause increased urinary excretion of potassium. Hypopotassemia renders the heart more sensitive to digitalis. Overdosage of the purified glycosides is often first evidenced by arrhythmias.

"The most important manifestation of digitalis intoxication is the occurrence of arrhythmias. Any type of arrhythmia and conduction disturbance may be caused by digitalis toxicity.

"The various arrhythmias caused by the irritant action of digitalis are atrial fibrillation, atrial flutter, or atrial tachycardia with block. . . . Ventricular premature systoles are also common manifestations of digitalis overdosage, particularly when digitalis and diuretics are used concomitantly. . . . Because both diuretics and myocardial infarction reduce the myocardial potassium, the heart becomes more sensitive than normal to digitalis. In these patients, bursts of multifocal premature beats are warning signs. . . . It is evident that the first treatment after withdrawal of the digitalis should be the administration of potassium. . . . It is best administered in solution orally, or intravenously. The absorption of enteric-coated tablets is unreliable.

"The chelating agent, trisodium ethylenediaminetetraacetic acid (EDTA), appears to be very effective in the treatment of digitalis toxicity.

"Magnesium, Dilantin, procaine amide or quinidine have also been found to be effective in the treatment of digitalis-induced arrhythmias. However, if conduction defects have resulted from depression of the myocardium, Pronestyl or quinidine may aggravate the condition because of increased block."

(Corday, Eliot, et al, American Heart Journal, Volume 46, pages 126-134, 1962)

"Your attention is called to the article by Dr. Charles Okey in this issue of the Maine Medical Journal concerning the diagnosis, dangers and need for adequate treatment of Group A streptococcal infections. It is hoped that full use will be made of available State Laboratory facilities. Further comment on streptococcal infections will appear in the next issue of this Journal."

*the first comprehensive  
regulator of  
female cyclic function*

# ENOVID<sup>®</sup>

(brand of norethynodrel with ethynylestrodiol 3-methyl ether)



*Simple adjustments of the dosage schedule with this versatile therapeutic agent enable the physician to: control dysfunctional uterine bleeding, regulate an abnormal menstrual cycle, enhance or suspend fertility, advance or postpone the menses, correct endometriosis often without surgery.*

**The Basic Action.** ENOVID (1) induces and maintains a pseudodecidual endometrium, preventing uterine bleeding, (2) inhibits pituitary gonadotropin, preventing ovulation.

When ENOVID is withdrawn, bleeding occurs in about three days and usually resembles a normal menstrual period in duration and volume of flow.

**Cyclic ENOVID Therapy.** When ENOVID is prescribed for 20 days of each cycle, commencing on day 5, the menstrual cycle will adjust to about 28 days regardless of menstrual timing prior to ENOVID therapy. A few cycles of therapy will frequently restore a normal pattern to women with irregularities as menorrhagia, metrorrhagia or secondary amenorrhea. Since ovulation is inhibited, ENOVID may be prescribed *cyclically* over prolonged periods to suspend fertility. During ENOVID therapy the ovary remains in a state of physiologic rest. After discontinuance of the drug the normal ovulatory pattern returns. Indeed, subsequent pregnancy appears to be enhanced through a probable "rebound" phenomenon. Thus, *cyclic* ENOVID administration has been successful in treating endocrine infertility.

**Continuous ENOVID Therapy.** When ENOVID is given on a *continuous* dosage basis, menstruation as well as ovulation is completely suspended. In endometriosis, *continuous* ENOVID therapy produces a pseudodecidual reaction with subsequent absorption of aberrant endometrial tissue. This often eliminates the need for radical surgery. When surgery is indicated, ENOVID is an effective adjunct preoperatively, as well as postoperatively, to prevent recurrence.

*Continuous* administration of ENOVID is also utilized in habitual abortion, providing bal-

anced hormonal support of the endometrium and permitting continuation of pregnancy.

**Emergency ENOVID Therapy.** In high doses, ENOVID has a prompt hemostatic effect and will usually control severe dysfunctional uterine bleeding within 6 to 24 hours. Prompt, high-dosage administration of ENOVID is also a rational recourse in threatened abortion.

**A Note on Safety.** The effects of ENOVID have been studied in more than 3,500 women during more than 49,500 menstrual cycles, representing 3,800 woman-years of experience. ENOVID has been administered *cyclically* to the same patients for as long as five and one-half years for ovulation inhibition without serious complication. For the present, however, ENOVID is not recommended for more than two years, although it is expected that this period will be lengthened as experience continues to accumulate. There has been no impairment of subsequent fertility and no effect on children born to women who conceived after discontinuing ENOVID therapy.

**The basic dosage** of ENOVID is 5 mg. daily in *cyclic* therapy, beginning on day 5 through day 24 (20 daily doses). Higher doses may be used to prevent or to control occasional "spotting" or breakthrough bleeding during ENOVID therapy or for rapid effect in the emergency treatment of dysfunctional uterine bleeding or threatened abortion.

ENOVID is available in tablets of 5 mg. and of 10 mg. Available on request: literature and references covering more than six years of intensive clinical study.

SEARLE

Research in the Service of Medicine  
G. D. Searle & Co., P.O. Box 5110, Chicago 80, Ill.

# Necrologies

SAMUEL R. WEBBER, M.D.

1895-1962



The International community of the St. Croix Valley, comprising parts of Washington County, Maine and Charlotte County, New Brunswick, was shocked and saddened to learn that one of its leading citizens and professional men, Samuel R. Webber, M.D. and his wife Marion, had been drowned in West Musquash Lake, while on a fishing holiday on Memorial Day.

To one who has been a very close colleague for over 30 years, it just doesn't seem possible that Sam and Marion have left us. It is only a few months ago that the St. Croix Medical Society gave them a party to welcome Marion to our midst and to present them with a token of our esteem and regard.

Dr. Samuel Webber was a leader among men and physicians, truly a doctor of the old school, following faithfully in the footsteps of his revered father. His skill and wisdom were always at the service of patient or colleague and his cheery manner and unfailing courtesy to all with whom he came in contact, made the day brighter for having seen him.

He graduated from Harvard Medical College in 1921, interned in surgery at the Peter Bent Brigham and started his practice in Calais, Maine in 1924 where his professional attainments were soon recognized on both sides of the border. He was several times President of both the Washington and Charlotte County Medical Societies, was a member of the executive committee and vice-president of the New Brunswick Medical Society, was on the Board of Censors of the Maine

Medical Association in 1948 and 1953, and greatest honor of all was his election as President of the New England Surgical Society in 1956 and '57. He was a Fellow of the American College of Surgeons and held a Certificate in Surgery in the Royal College of Physicians and Surgeons of Canada. He was a member of both the American and Canadian Medical Associations, Chief of Surgery at the Charlotte County Hospital and Chief of Staff at the Calais Regional Hospital.

He was a good woodcock shot, a keen dry fly fisherman, a golfer and a great reader, as his excellent library showed.

Beloved and respected by all, he will be sadly missed by friends and patients on both sides of the border. Over his resting place could so well be graven the words "Here lies a very gallant gentleman."

H.S.E.

DEFOREST WEEKS, M.D.

1890-1962

Doctor DeForest Weeks came from a medical family. His father, a great man, was an old time general practitioner in Cornish, Maine, and cared for folks for miles around. His father's cousin was Dr. Stephen H. Weeks, at one time Professor of Anatomy and later Professor of Surgery at the Medical School of Maine. In his time he was the leading surgeon in Maine. His house stood on the corner of Congress and State Streets in Portland and is now used by the Nursing Department of the Mercy Hospital.

Doctor Weeks, after attending the schools in Cornish, was graduated from Bowdoin College in 1911, and from Bowdoin Medical School in 1914. He interned at the Maine General Hospital where he met his future wife then Miss Addie Wagner, the most capable nurse in her class and student head nurse in the operating room. Soon after he began his practice in the Woodfords section of Portland, he entered the Army Medical Corps as a Lieutenant, serving in 1918 and 1919. His oldest child, Elizabeth, was born while he was overseas.

On his return, he again practiced in Woodfords. Before the change over in the staff at the Maine General Hospital, he was a junior on the surgical service. For a time he was an examiner for the Veteran's Bureau. In the course of years, as his practice grew, he was a member of the staffs of the Maine Eye and Ear Infirmary, the Maine General Hospital and later the Maine Medical Center, the St. Barnabas Hospital, the State Street Hospital and the Mercy Hospital. He was at one time City Physician.

As a physician, he was kindly, industrious and unswervingly honest and honorable and built up a large general practice.

Doctor DeForest Weeks was 72 years of age and had practiced in Portland for 48 years. He died suddenly Sunday morning June 10, 1962, in a Portland hospital after a brief illness.

Doctor Weeks was born April 1, 1890, in Limington, son of Doctor George W. and Stella Libby Weeks. He served on the School Board of Portland, was a 32nd Degree Mason, a past president of the Executives Club and of the Fraternity Club. He was a member of the Cumberland County Medical Society, the Maine Medical Association, the American Medical Association, the Woodfords Club, and was a former deacon of the Woodfords Congregational Church. He is survived by his widow, a son George DeForest Weeks, of Appleton, Wisconsin; two daughters, Mrs. Elizabeth W. Tapia of Rochester, New York and Mrs. Estelle W. Wellman of Sudbury, Massachusetts; six grandsons; and a sister, Mrs. Marguerite Johnson of Cornish.

# County Society Notes

## ANDROSCOGGIN

A meeting of the Androscoggin County Medical Association was held at St. Mary's General Hospital in Lewiston, Maine on September 20, 1962 with 29 members present.

Resolutions on the death of Frank P. Methot, M.D. were read and it was resolved that a copy be spread on our records and a copy sent to the immediate family.

There was considerable discussion concerning the Sabin oral vaccine program, and the following members were appointed by the President, George B. O'Connell, M.D., to act as liaison committee between the society and local Health Officers: Henry C. Thacher, M.D., Chairman, Auburn; Horacio A. Lichter, M.D. and Robert D. Wakefield, M.D., both of Lewiston. Daniel R. Shields, M.D. presented a report on the status of Blue Cross and Blue Shield for the aged.

The Scientific Program consisted of a symposium on "Uses of Isotopes in Current Medical Practice" with the following participants: Lawrence A. Nadeau, M.D., John T. Konecki, M.D., Paul J. Fortier, M.D., John Milazzo, M.D., Milan A. Chapin, M.D., Charles A. Hannigan, M.D. and Richard Goldman, M.D.

A question and answer period followed and the panel was thanked for their short, concise presentation.

DONALD L. ANDERSON, M.D.  
*Secretary*

## OXFORD

The annual meeting of the Oxford County Medical Society was held at the Bethel Inn in Bethel, Maine on October 3, 1962.

Charles W. Eastman, M.D. of Livermore Falls, Councilor for the Second District, was a guest.

The following slate of officers were elected for 1963:

President, John Young, M.D., Bethel

Vice-President, Leonidas B. Kudisch, M.D., Rumford

Secretary-Treasurer, Albert P. Royal, Jr., M.D., Rumford

Following an excellent dinner, a concert and demonstration of the Hammond organ by Joseph Zinni was enjoyed by 39 members and their guests.

ALBERT P. ROYAL, JR., M.D.  
*Secretary*

## New Members

### AROOSTOOK

Ernest D'Agostino, M.D., 45 Beechwood Avenue, Port Washington, New York

Eric F. Nicholas, M.D., Fort Fairfield

G. Douglass Timms, M.D., Main Street, Mars Hill

## Letter To The Editor

Daniel F. Hanley, M.D., Editor  
Journal of the Maine Medical Association

Dear Doctor Hanley:

Away back in 1938, I wrote a paper,\* trying to link cigarette smoking to lung cancer. It has, therefore, disturbed me greatly ever since to know that smokers are not yet aware, that if they continue to smoke, they are likely to die of lung cancer. Since 1938, many M.D.'s the world over have expressed opinions similar to mine, but with too little effect in reducing the smoking of cigarettes.

The smoking of cigarettes is increasing, rather than lessening, because what we M.D.'s say, who know its dangers, is so effectively counteracted by the propaganda put out by the commercial interests, who, for other reasons, have other beliefs. I am glad the Maine Medical Society last week passed a resolution in which, it said "It is estimated that one million of our present population of school children will die of lung cancer, if present cigarette smoking trends continue." I am quite concerned by the persistent, well-done promotion of cigarette smoking by T. V., radio, newspapers and magazines.

It was my privilege to be one of six students, who daily studied Surgery under Dr. Evarts Graham at Washington University, during my senior year in the Medical School there.

As everyone who has followed Surgical History knows, Dr. Evarts Graham was the first man to successfully remove a human lung. I was in St. Louis, when this was done. It was a

great step forward in Surgery, for it meant the saving of many lives of those afflicted with lung cancer, lung abscess and other serious diseases of the lungs. Just after World War I, as a patient, I spent over two years in the chest wards of army hospitals.

Dr. Graham's technique for the removal of a human lung has since been improved and performed by many others, but it is *not* the most effective way to treat lung cancer.

The most successful way to control lung cancer and an inexpensive and effective way is to simply stop smoking. The fact that I have been saying that for thirty years with so little effect amazes me. Many others methods of controlling cancer in the various organs of our bodies are effective, but none are more effective than simply stopping smoking. Though more people now than ever before know that stopping smoking will control cancer in no small way, each day more people, especially teenagers begin the unfortunate, vicious, dirty and dangerous habit. It is like any other addiction, once it becomes a fixed habit.

Right after Dr. Graham first removed a man's lung, I began to take a cigarette history on all patients who were diagnosed cancer of the lung. I took these histories during my five years of postgraduate study in the Bernard Free Skin and Cancer Hospital of St. Louis and the New York Skin and Cancer Hospital and elsewhere. Therefore, even before 1938, I was completely convinced that cigarette smoking and lung cancer and heart disease were definitely related. In 1938, I said it in my weekly column. I took these cigarette histories with great interest, for I have never smoked and I had long believed that smoking and lung cancer were related, though no good statistical proof was yet available. I knew that cancer of the tongue and mouth were linked to the smoking of cigars,

\*"Why Smoke" appeared in the January 14, 1938 issue of "The Church World."

cigarettes or pipes, so I thought that cancer of the lungs might be linked to cigarette smoking, which, even in my grade school days, I had been told were "coffin nails." As I have said, I first published my not too well supported beliefs that smoking is a cause of lung cancer in 1938, and as many know, I have reiterated this conviction often in the intervening years, publicly and privately.

Among the supporters of the smoking habit are those who think that they lack the will power to stop. This group constantly raises its voice in defense of the habit. They are really trying to defend themselves. They try to rationalize themselves into believing that the thing they want to do, is the right thing to do and a safe habit to have. They feel with no good justification: It is the "other fellow," who may die of lung cancer, but such a catastrophe will not hit me. They tell themselves that it has not been proven that cigarette smokers are more likely to die of cancer of the lung, than those who do not smoke.

Among the other supporters of the smoking habit is the indifferent group, the "don't care" people, who feel more or less hopeful about everything; in other words, the fatalists. Then there are the "busy" people, who feel that they need the "relaxation" or the "pleasure." There is also the uninformed group, who know little of the dangers involved and who foolishly continue to smoke.

All of these people are played upon by the tremendous, all-pervading commercial interests, who work for personal profit. They have millions of dollars to spend and they use every mass propaganda media available to prove to the public with alluring and convincing advertisements that the research men and the doctors are all wrong and that smoking is O. K. All who read or listen are daily being brain-washed.

All of these devotees of the smoking habit are doing a tremendous damage to the young people of this generation by their personal example and their spoken word.

That my years of preaching against smoking has had so

little effect is one of the disappointing experiences in my many years of trying to be a dedicated doctor. The dedicated physician puts the interest of his patients first, not his own pocket-book, and he is bitterly disappointed when what he knows to be true is not accepted by patients who get viewpoints from others who are less well-informed medically and whose interests are not so unselfish.

Though there has been a great inability on the part of many smokers to stop smoking, I am glad to say that many physicians have been able to do so. They stop because they know the cancer-producing effects better than their patients do. They know, too, that death from cancer of the lung is a terrible death and only a very small percentage are saved by the removal of one lung, in order to save the patient. Frequently, both lungs are affected. It is such a tragedy, when a patient dies of a disease he or she could have prevented by simply cutting out a bad habit, like smoking. I believe smoking should be called an addiction, for it is that in most smokers.

I am of Holland Dutch blood 100% and true to my inheritance, I keep believing and saying that young and old must be convinced that cigarette smoking is harmful and that a sure way to save lives is to simply stop smoking. "The life you save may be your own." We Dutch have tenacity and perseverance — even when we seem to make no progress, and our stand is unpopular and unconvincing, so I keep repeating this, and my other deep conviction, which I began preaching twenty-five years ago, when on the National Staff of the American Red Cross: "Slow down and live," and "slower driving means a lower death rate."

Dr. Graham himself died of cancer of the lungs. "He saved others, himself he could not save." He stopped smoking as soon as he was sure it was a dangerous habit. He did everything in his power to help to save his fellow man. Can we do less?

Adrian H. Scolten, M.D.  
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# The Journal of the Maine Medical Association

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## Malignant Melanoma In Eastern Maine

THOMAS H. PALMER, JR., M.D.

The purpose of this paper is to record ninety-one cases of malignant melanoma encountered over a period of fourteen years in Eastern Maine, and to compare the characteristics and treatment of melanoma in this geographic area with large reported series in the literature.

### MATERIAL

During the years 1947-1960 the diagnosis of malignant melanoma was made by microscopic examination in ninety-one cases at Eastern Maine General Hospital, exclusive of melanoma of the eye, which has somewhat different characteristics and will not be discussed here. In all cases the diagnosis was made by competent pathologists and all questionable cases were studied by at least two pathologists. The pathology department of the hospital receives tissue specimens from many smaller hospitals and from a large number of physicians throughout Eastern Maine. Clinical and follow up data were obtained from hospital records and from the patients' physicians.

### AGE AND SEX

The youngest patient in this series was thirteen and the oldest was ninety-one at the time diagnosis was made. Allen found that 63% of nine hundred and thirty-four patients with melanoma were between the ages of thirty-one and sixty, an age incidence similar to that of many other carcinomas.<sup>1</sup> In the present series only 37% of the patients were in this age group, with a much higher percentage in the older decades (Table 1). This may be a reflection of the well known fact that Maine has a higher proportion of elderly people in the population than most other states.

As in other reported series, the incidence of melanoma was approximately equally divided between the

TABLE 1.

MALIGNANT MELANOMA — E. M. G. H.	
Age	Number of Patients
13 - 20	4
21 - 30	12
31 - 40	9
41 - 50	14
51 - 60	10
61 - 70	20
Over 70	20
Not Known	2

sexes.<sup>1</sup> There were forty-eight males and forty-three females. It is of interest to note that the prognosis of melanoma in females is twice as good as in males.<sup>1</sup> It has been believed in the past that pregnancy has an adverse affect on melanoma, and one patient in this series underwent therapeutic abortion. However, it has been demonstrated recently that pregnancy plays no role in the prognosis of melanoma.<sup>4</sup>

### LOCATION OF PRIMARY LESION

In the present series melanoma of the head and neck region occurred equally as frequently as that of the extremities (Table 2). This differs from other series in which the extremities are by far the most common site.<sup>1,3</sup> In two patients the primary site could not be determined.

### TREATMENT

#### Prophylaxis

It is generally agreed that the majority of malignant

TABLE 2.

MALIGNANT MELANOMA — E. M. G. H.		
<i>Location of Primary Lesion</i>		
	<i>Number</i>	<i>Percent</i>
Head & Neck	32	35%
Trunk	21	23%
Extremities	32	35%
Genital	4	4%
Undetermined	2	2%

TABLE 3.

MALIGNANT MELANOMA — E. M. G. H.	
	<i>Treatment</i>
91 Cases	1947 - 1960
80 Cases	Local Treatment
22 Cases	Regional Node Dissection
11 Cases	Palliative Treatment Only

melanomas develop from preexisting moles, although they may arise de novo. It is recommended that all moles be excised in areas that are subject to chronic irritation, such as the palms, the feet, at the belt line, or beneath shoulder straps and collars.

*Local excision*

Proven melanomas should be widely excised. Removal of the lesion with a three or four centimeter margin of normal skin, and the underlying fat and deep fascia is considered satisfactory.<sup>5</sup> Very often a skin graft is necessary to close the remaining defect. Before radical local excision is undertaken, there should be proof that the lesion is a malignant melanoma. Such benign lesions as seborrheic keratosis or pigmented nevus with infection can be mistaken clinically for melanoma not infrequently. The size and location of the lesion are important regarding approach to establishment of the diagnosis. Total biopsy should be done when this is feasible. Simple biopsy of very large lesions, or those on the face or digits is sometimes required.

In the present series eleven of the ninety-one patients (12%) had far advanced melanoma when the diagnosis was made, and received only palliative treatment (Table 3). The majority of patients underwent wide local excision of the primary lesion. However, in some cases, excision of a melanoma with a skin margin only a few millimeters in width and a deep margin barely through the skin was accepted as definitive local treatment. It is regrettable to find that two lesions were fulgurated and three lesions were treated by x-ray in physicians' offices without biopsy. Three lesions were excised and discarded without microscopic examination. All eight of these patients died of widespread metastatic melanoma. Not all of these mismanaged cases were treated in the early years of this study.

*Lymph node dissections*

There is general agreement that regional lymph node dissection should be carried out when there are clinically suspicious lymph nodes present, without demonstrable distant metastases. However, the question of removal of the regional lymph nodes when there is no clinical evidence of involvement is somewhat controversial. Many authors have reported that 20-50% of patients who underwent so-called prophylactic regional node dissections were found to have microscopic evidence of metastasis in one or more nodes.<sup>2,6,7</sup> This is a rather powerful argument in favor of prophylactic node dissection.

Preston<sup>8</sup> has found that 42% of patients who developed recurrent melanoma after local excision had the recurrence in regional lymph nodes. Lane<sup>5</sup> has reported that the five year survival rate of patients who have lymph node metastasis only by microscopic examination is 60%, compared with 10% in patients with clinically positive lymph node metastasis.

It is therefore recommended that in the absence of distant metastases all patients with proven melanoma should undergo excision of the regional lymph nodes, with two exceptions. When the primary lesion is so located topographically that its lymphatic drainage is to more than one lymph node station, prophylactic node dissection would be too extensive to be feasible. A melanoma of the trunk close to the midline, for example, may spread bilaterally in the lymphatics. The second exception is a very superficial melanoma, with microscopic demonstration of only epidermal and junctional involvement. These lesions have a very good prognosis with local excision alone.<sup>5</sup>

Of eighty patients in this series who were treated with the possibility of cure, twenty-two underwent regional node dissection. There were no operative deaths. It is notable that node dissections were done much more frequently in recent years. More than half of these node dissections were done in the last five years of the fourteen year study. In the early years of the study patients were frequently referred to medical centers in Boston for node dissection, whereas more recently the operations have been done at hospitals in the patients' home area.

*Chemotherapy*

In recent years some of the patients in this series have been treated with various chemotherapeutic agents, usually after surgical treatment had failed, but sometimes in conjunction with surgery. It is not within the scope of this paper to discuss the use of this interesting and hopeful mode of therapy.

RESULTS

There is a tendency among some physicians to adopt a despairing attitude toward malignant melanoma. Actually the prognosis is not as bad as many believe — (Table 4), and the results are comparable to or better than those of some other carcinomas such as those of

TABLE 4.

MALIGNANT MELANOMA

5 Year Survival Rate

Treated

Author	Year	Cases	% Survivors
de Cholnoky	1941	81	42.3
Sylvén	1949	291	30.8
Raven	1950	72	9.7
Pack et al	1952	575	21.4
Hall et al	1952	132	28.0
Stewart et al	1953	78	24.3
Preston et al	1954	164	15.4
Catlin	1954	80	36.0
Lund & Ihnen	1955	73	26.0
Brandt	1956	112	20.5
Meyer	1957	107	51.0
Royster & Baker	1957	66	39.0
Lane et al	1957	105	34.3

(From *Journal of Cancer*, 1958, Page 1025)

TABLE 5.

MALIGNANT MELANOMA — E. M. G. H.

Results

51 Cases Treated Over 5 Years
17 Cases (33 1/3%) Living and Well
26 Cases Died of Melanoma
3 Cases Died of Unrelated Cause
2 Cases Living With Recurrence
3 Cases Untraced

the stomach, lung, or breast. It is safe to say that the physicians who treated melanomas in this series by minimal local excision, and excised, fulgurated, or radiated moles without microscopic examination, would never treat a breast lesion in this fashion and would certainly adopt a much more aggressive attack on a neoplasm of the lung or stomach. It is true that some melanomas metastasize widely at an early stage, but this is often not the case, and an important role is played by the presently imponderable factors of host resistance and tumor virulence. Since one cannot determine which melanomas will respond favorably to treatment they should all be attacked vigorously.

It has been established that approximately two thirds of the patients who develop recurrence of melanoma do so within two years, and four fifths of the recurrences are within five years.<sup>5</sup> In this series of ninety-one patients, fifty-one were treated five or more years before the time of study (Table 5). Of these fifty-one patients, seventeen or 33 1/3% were alive without evidence of recurrence. Twenty-six patients died of melanoma and three died of other causes. Two pa-

tients were alive with recurrence at the time of study. Three patients were untraced.

Thirty-two of the fifty-one patients had only local treatment of melanoma, and eleven of these were living and well. Ten patients underwent regional node dissection and six were living and well. Although the five year survival rate is much better in those patients who underwent node dissection, the number of patients is too small to be of significance. Seven patients had no treatment, or only palliative treatment, because of the advanced state of their disease at the time that it was diagnosed. Forty per cent of those who were treated with a possibility of cure were alive and well after five years.

Fourteen patients were treated between three and five years prior to the time of study. Eight of this number were living and well, two had known recurrence, and four had died of their disease. Seven of the fourteen patients underwent regional node dissection, six had local treatment alone, and one patient had advanced melanoma when the diagnosis was made.

SUMMARY

Ninety-one cases of malignant melanoma have been diagnosed at the Eastern Maine General Hospital in a period of fourteen years. The age incidence was considerably older than in other reported series. Melanoma of the head and neck was more common than usual in this series. In more than a few instances management of the primary lesion was not in conformity with accepted standards. Regional node dissections have been carried out with increasing frequency. The overall five year survival rate was 33 1/3%. Forty per cent of those patients who received other than palliative treatment were alive and well after five years.

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# Aseptic Necrosis Of Femoral Heads Associated With Steroid Therapy

CARL W. RUHLIN, M.D.\*

Bilateral aseptic necrosis of the femoral heads has been studied and associated with several clinical entities. The etiology of the necrosis is not well understood, even in the most widely studied series of cases such as the clinical entity known as Legge Perthe's disease. Dubois and Cozen<sup>1</sup> have done extensive studies on bilateral avascular (aseptic) necrosis associated with Systemic Lupus Erythematosus. Ratcliffe and Wolfe<sup>2</sup> have offered a clinical study of avascular necrosis of femoral heads associated with Sick cell traits.

The studies of Dubois and Cozen revealed no association between pain in the hip joints and corticosteroid therapy. The studies did reveal the fact that the weight bearing joints were most frequently involved. Knee joints as well as hip joints were involved in the process of aseptic (avascular) necrosis. The hip joints involved showing aseptic (avascular) necrosis revealed a complete cycle of subchondral necrosis, revascularization and repair. There was typical sclerosing of the weight bearing surface of the femoral head and approximating surface of the acetabulum.

The biopsied specimens of the aseptic femoral heads revealed a thrombosis of the terminal vessels as confirmed both in cases associated with Systemic Lupus Erythematosus and Sick cell traits.

## CASE REPORT

The single case of aseptic necrosis of the femoral heads associated with steroid therapy studied at the Eastern Maine General Hospital was in a seventeen year old female. The case was admitted for study because of a moderately severe bilateral limp associated with constant pain and loss of motion of the hip joints. The history was complicated as the patient was suspected of having rheumatoid arthritis treated by prolonged steroid therapy. There had been two previous hospital admissions before her entrance to the Eastern Maine General Hospital.

The patient was struck on the left shoulder with a baseball in 1959. The left shoulder pain was associated with pain in the left elbow and both knees. At the time of this initial hospitalization the patient was found to have a systolic murmur in the pulmonic area. Rheumatic fever was suspected and the patient was placed on penicillin and steroids, (Decadron®). On this hospital admission no mention was made of pain in the region of the hip joints.

The patient remained in the hospital four (4) months at which time X-rays were taken of the painful joints and chest. The chest plate was normal and the films of the joints revealed no evidence of trauma or disease. The patient during her hospital stay was improved under bed rest, penicillin and

steroid therapy. The patient was discharged as improved.

Two months following the cessation of all therapy the shoulder pain returned and the patient was readmitted to the hospital in October, 1960 with a diagnosis of pain, recurrent, associated with rheumatic fever. The "moon faced" appearance of the patient was noted on this second admission. The patient also had a noticeable cough. The sedimentation test was increased. X-rays of the chest revealed normal lung and cardiac fields.

The patient was placed on steroid therapy and bed rest. A complaint of pain in the left mandible region was recorded and dental consultation requested. Consultation revealed no evidence of trauma or osteomyelitis. There were many dental caries with absorption and crown destruction. The teeth were extracted. Previous to the extractions no improvement was found in her condition. Two electro cardiograms were suggestive of myocarditis but not conclusive. Cardiac fluoroscopy was repeated revealing normal excursion, rhythm and size. Repeated urine and blood counts were normal with the exception of the white count which was elevated. The sedimentation rate remained elevated.

Following the extraction of the teeth the pain subsided in all joints, blood counts and the sedimentation rate became

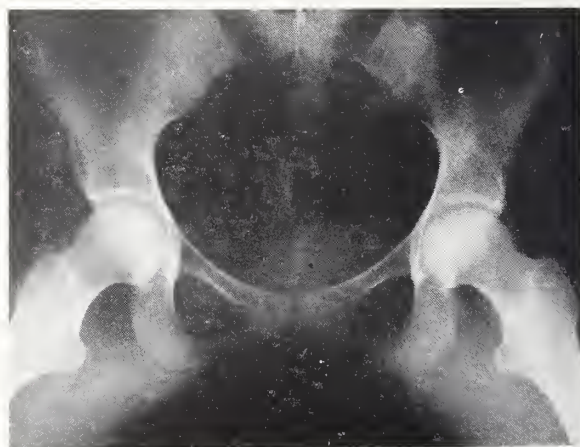


FIG. 1-A



FIG. 1-B

\* From the Orthopedic Service, Eastern Maine General Hospital, Bangor, Maine.

normal. Mandibular pain also subsided and the patient was discharged from her second admission as improved. A return date for check up examination in two months was suggested but the patient failed to keep the appointment. The patient gained forty (40) pounds during her hospital stay. When she left the hospital all medication was stopped.

Nine months following the second hospital discharge the patient was examined at clinic because of her complaint of pain in the region of both hip joints. X-rays revealed normal symmetrical hip joints with no evidence of trauma or disease. There was, at this examination, no evidence of "moon face," pain in shoulders or knees. It was noted, however, that the breasts, abdomen and thighs were covered with large blue striae. The patient had lost the forty (40) pounds she had gained during her second hospital stay. The patient was again examined in two months still complaining of pain in the hip joint area. Blood counts and sedimentation rate was normal. The pulmonic murmur could not now be heard.

The patient was admitted to the Eastern Maine General Hospital fourteen (14) months following her second hospital admission because of the continued complaint of hip joint pain. For the first time x-rays revealed the probable cause of pain in the hip joint regions. The chief x-ray findings were located in the head of both femora. There were mottled areas of decreased density producing some irregularity of the bony articular surface, associated with generalized increased density of the heads of both femora. Both joint spaces were slightly narrower than usual yet both were symmetrical. There was no destruction of the joint capsules. (Fig 1.)

At the time of admission to the Eastern Maine General Hospital clinical examination revealed the previously mentioned body striae. The hip joints showed considerable restriction of motion in all directions. Abduction and external rotation were particularly painful. All other major joints of the upper and lower extremities were normal. Pelvic examination revealed a nulliparous cervix and it was determined that the striae were not due to pregnancy. Electrocardiogram revealed normal heart tracing in all leads. Clinical examination

revealed normal heart and lungs. These findings were confirmed by fluoroscopy. All laboratory tests proved to be normal with the exception of elevation of the total cholesterol.

Urine — Normal	Sed. time —	60 min.
Blood — Hb 12 gms. 77%	Sed. Index	17 min.
WBC — 5,250	Uria Nitrogen	16
Neutro 67	Uric acid	5.7
Easin 1	Total Protein	70 gms.
Lymphs 29	Total Cholesterol	328 gms.
Monocytes 3	Alkaline	
	Phosphotase	3
	Phosphorous	4.3 mgs.
	L.E. Cells	None on 3 tests
	R.A. Test	Negative

SUMMARY

The case presented is that of a seventeen year old female with body striae and bilateral symmetrical aseptic necrosis of the heads of the femora associated with prolonged intensive steroid therapy.

Studies failed to support the diagnosis of Rheumatic Fever, Rheumatoid Arthritis or Septemic Lupus Erythematosus.

Subsequent follow up for eight months fail to reveal any change in the condition of the hip joint or striae.

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205 French Street, Bangor, Maine

Drug Research Increased

Despite the harassment of a prolonged investigation often characterized by sensationalism, the pharmaceutical industry spent 13 per cent more on research and development in 1961 than in the year previous. The pharmaceutical industry's investment in research is now three times that of average industry. In the period from 1959 to 1961 nearly 500 scientists were added to the staffs of 23 larger companies . . . The industry's faith in its competence is well founded on past performance. It will be supported by those it serves. — Editorial in *New York State Journal of Medicine*, Sept. 1, 1962.

# Vertebral Osteomyelitis — A Case Report

JAMES J. BROD, M.D.

Vertebral osteomyelitis is commonly mistaken for a visceral lesion, with consequent delay of some weeks in the diagnosis. X-ray evidence of bone infection appears one to two weeks after the onset of the disease at the earliest, and may be much later when the disease is modified by antibiotics. That the disease is an acute infection is usually obvious from the outset, its localization may be obvious only in retrospect. The case presented illustrates the difficulty of diagnosing this infrequent disease.

## CASE REPORT

A 72-year-old woodsman, with no significant past illnesses, was in rugged good health until early November 1961. He awoke with chills and fever one morning, was treated with penicillin, and because of the lack of response was hospitalized. In three days a positive diagnosis could not be established, and the man had become desperately ill. He was transferred to the Eastern Maine General Hospital, presumed to have urinary obstruction because of his left flank and lower abdominal pain.

He was examined by a urologist on admission, and found to have slight enlargement of the prostate, minimal pyuria (4-6 WBC/hpf), and only four ounces of clear residual urine. There was not sufficient tenderness in his flank to explain the grave toxic picture — a patient with a temperature of 104 degrees and noticeably irrational. An emergency medical consultation was obtained. The only complaint which could be elicited at that time was generalized lameness. His temperature was 104 degrees, respiratory rate 26, and pulse 104. Physical examination revealed occasional rales at the left base. His abdomen was slightly distended. There was tenderness in the left mid-abdomen, and definite left CVA tenderness. The man had a large left scrotal mass presumed to be a hydrocele. There were no other significant findings. A chest x-ray showed an area of consolidation in the left lower lobe. WBC was 12,000 with 89% polys. A blood culture was obtained, and the patient placed on antibiotics as a life-saving measure.

The patient was treated with the tentative diagnosis of left lower lobe pneumonia, but the medical consultant at that time noted that the symptomatology, physical findings, and radiographic evidence were inconsistent with the duration and severity of the sepsis. He felt at that time that the patient should be considered to have a fever of unknown origin. The emergency antibiotic regime of three grams of Chloromycetin and 2,500,000 units of penicillin daily was continued for ten days. After two days he was much improved, and an intravenous pyelogram was done showing no abnormality. In four days he was afebrile, and gotten out of bed. He was ambulant thereafter with no complaint of pain. A chest x-ray on his twelfth day showed complete resolution of the pneumonic process, and he was discharged on the following day.

The blood culture had grown out *B. coli* highly sensitive to Chloromycetin. His urine culture had also grown out *B. coli*, staph aureus, and a rare strep.

He was followed in the out-patient department regarding his prostatitis, a factor which was felt to be of questionable significance in his acute febrile illness. For three weeks following discharge he had intermittent pain in both groins which

radiated up over the hips and into his back. He was seen in the urology clinic where sounds were passed and his prostate massaged. After this visit his symptoms worsened, and he complained of a lot of soreness "across the top of his hips." The pain became quite severe and made it difficult for him to move around. He had only very slight relief of pain with aspirin. He felt that he had lost a good deal of weight.

He was readmitted on 12/27/61 because of the above clinical picture. Examination at that time showed bilateral CVA tenderness, more marked on the left. Again there were findings of mild prostatic disease. His temperature on admission was 101.4 degrees, but he was almost afebrile thereafter. He had no leukocytosis and no residual urine. An orthopedic consultation was requested on 12/30/61 to explain his back pain. On orthopedic examination his maximal tenderness was localized to the left of L<sub>3</sub>. X-rays of the lumbar spine were negative. It was felt that his illness was due to a septic process, probably in the retroperitoneal space to the left of L<sub>3</sub>. Vertebral osteomyelitis was considered, and it was suggested that further time must elapse to rule out this disease. Chest x-ray at this time showed a minimal fibrotic residue of the old pneumonic process. An intravenous pyelogram was again normal except for evidence of a slightly enlarged prostate elevating the base of the bladder. During early January the patient was repeatedly re-examined and multiple x-rays and lab studies were done. Notwithstanding, a definitive diagnosis was not made.

There had been a trace of sugar in his urine on admission, and one week later there was 1.65%. His mild diabetic state was treated without complication thereafter with Orinase. On admission his hematocrit was 35%, and there was no leukocytosis. The WBC rose to 13,000 one week following admission, and his sedimentation rate at that time was 26 mm./hour. There was still no significant fever. Blood cultures were negative. A systolic heart murmur was heard by a cardiologist on 1/8/62, and because it had not been noted by previous observers the diagnosis of sub-acute bacterial endocarditis was strongly considered.

All of his x-rays were reviewed, and the radiologist questioned the significance of a soft tissue shadow at the level of T<sub>12</sub> seen on one of the films of his GI series. He was re-examined clinically, and the patient either had developed distinct spinal tenderness at the dorsal lumbar junction, or the author had failed to elicit this sign on examination two weeks previously. X-rays of this area were obtained, and there was evidence of marked destruction of the T<sub>11</sub>-T<sub>12</sub> interspace with related bone destruction, peripheral sclerosis, and a marked soft tissue mass. Figure 1 shows these x-rays which left little doubt that we were dealing with a pyogenic vertebral osteomyelitis.

*Staphylococcus aureus* is the usual etiologic agent,<sup>1</sup> but the previous blood culture cast doubt on this. After interservice consultation it was felt that the possibility of making an exact bacteriological diagnosis outweighed the potential hazards of a vertebral body biopsy, and after the necessary instruments were obtained, a needle biopsy according to the technique of Craig<sup>2</sup> was done. Figure 2 shows the localization of the needle when the biopsy was obtained.

Culture of the lesion showed *B. coli* which, to our surprise, was still highly sensitive to Chloromycetin. Microscopic examination showed no evidence of tuberculous granuloma,



FIG. 1-A



FIG. 1-B



FIG. 2-A

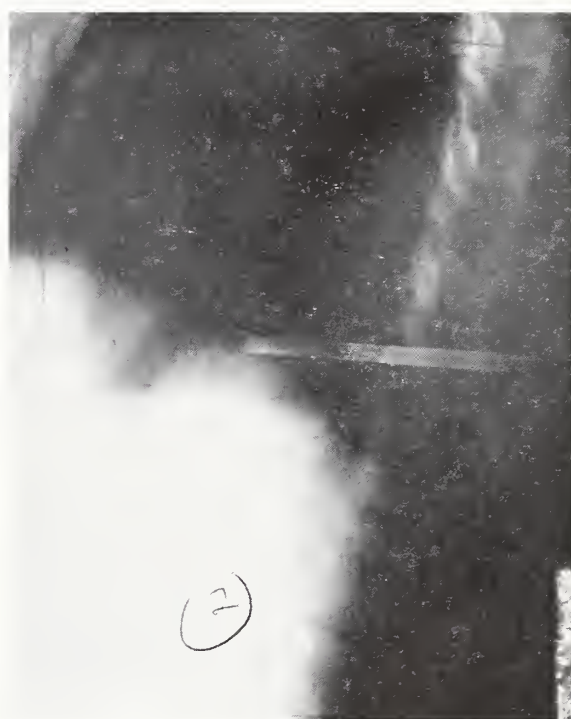


FIG. 2-B

and acid-fast stains were negative. The organism was also highly sensitive to tetracycline, and since Chloromycetin had failed to eradicate the infection in the acute stage, it was elected to treat the osteomyelitis with this drug. On the advice of Dr. Paul Beeson of Yale University,<sup>3</sup> streptomycin was added to the regime; he felt that, "as with the enterococcus, it (streptomycin) may show a synergistic effect when combined with another agent." This was done despite the in vitro finding of resistance to streptomycin. He was treated with tetracycline, two grams a day for two weeks, and one gram a day for two months. Two grams of streptomycin were given for ten days, and then one gram daily for two weeks.

The patient tolerated the biopsy well, and was placed in a plaster jacket from sternum to knees. He was asymptomatic

and steadily improved thereafter. He was discharged on 2/15/62, and kept on complete bed rest at home.

On 4/17/62 he had gained so much weight that he was suffering from the tight cast. He was readmitted and his cast changed. At this time his sedimentation rate was 10 mm. per hour. X-rays showed very satisfactory progression of healing. His antibiotics were stopped, and the new plaster jacket did not include pants. He returned home, and bed rest was continued. X-ray on 7/2/62 showed further healing, and clinical examination at this time showed no sign of recrudescence. He admitted at this time that he had been doing a small amount of walking at home. The cast was bivalved and strapped, and because of his economic circumstances, used in lieu of a brace. He was shown hyperextension exercises, and advised to gradually spend more and more time



FIG. 3

out of the cast. X-rays on 8/27/62 showed further healing, and again there was no pain or tenderness in his back. He was advised to do light work only this winter. It was somewhat disappointing to find no evidence whatsoever of interbody fusion at this time, Figure 3.

#### COMMENT

The case presented records the difficulties of diagnosing vertebral osteomyelitis even when one is searching for it. Elevated sedimentation rate and persistent back pain, worse in the upright position, are characteristics of this disease. It is embarrassing to report how badly misled we were by what, in retrospect was radiating pain in the T<sub>12</sub> distribution.

The advanced destructive changes present on 1/15/62 indicate that his entire illness starting in November 1961 was vertebral osteomyelitis. A very substantial course of Chloromycetin only modified this disease, it certainly did not cure it. This modified course of osteomyelitis has become quite common in the antibiotic era. While the microscopic structure of bone severely prejudices the effectiveness of any systemic agent, as shown by this case, it does not preclude a beneficial effect. Without antibiotics it is hard to believe that

there would have been clinical arrest of this man's disease in the absence of boney fusion.

The duration of antibiotic treatment of bone infection is a difficult question to discuss. A recent article authorized by the American Medical Association Council on drugs<sup>4</sup> offers the following statement: "In patients showing steady improvement treatment with these (antibacterial) agents should be continued for a minimum of three weeks and sometimes for as long as six weeks." As recorded above, our inclination was towards a longer period of treatment. We considered the use of gamma globulin, but felt that the favorable prognosis currently reported with conventional methods<sup>5</sup> indicated that this expensive modality be reserved for use in the event of failure.

It is interesting to relate this man's disease to a number of reported cases of vertebral osteomyelitis due to *B. coli*.<sup>6</sup> In these cases the infection is thought to spread by the vertebral veins of Batson, and there is frequently a history of urologic manipulation. This man has a history of urologic manipulation, but the onset of the disease apparently antedates this manipulation.

#### SUMMARY

A case of vertebral osteomyelitis due to *B. coli* is reported. The disease appears to have arisen from the urinary tract without history of manipulation. A precise bacteriological diagnosis by vertebral body needle biopsy proved of value. Diagnosis and treatment are briefly discussed.

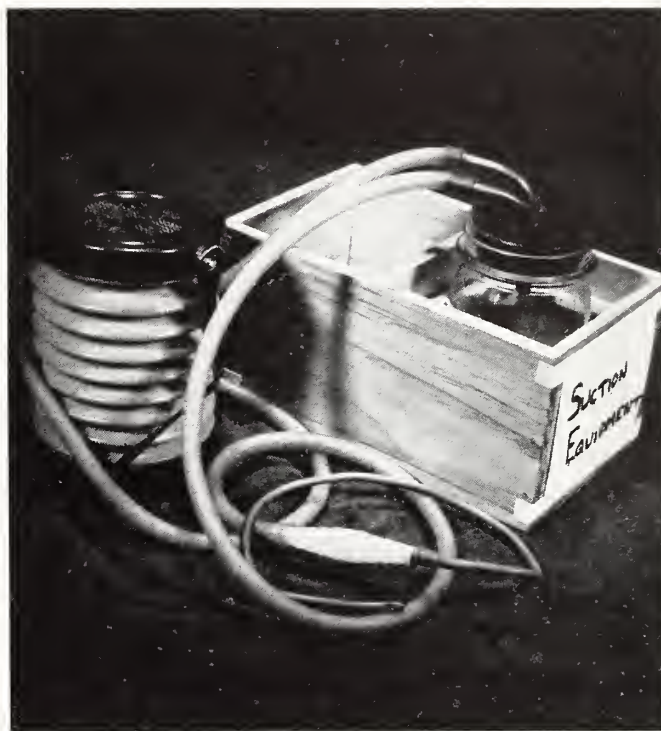
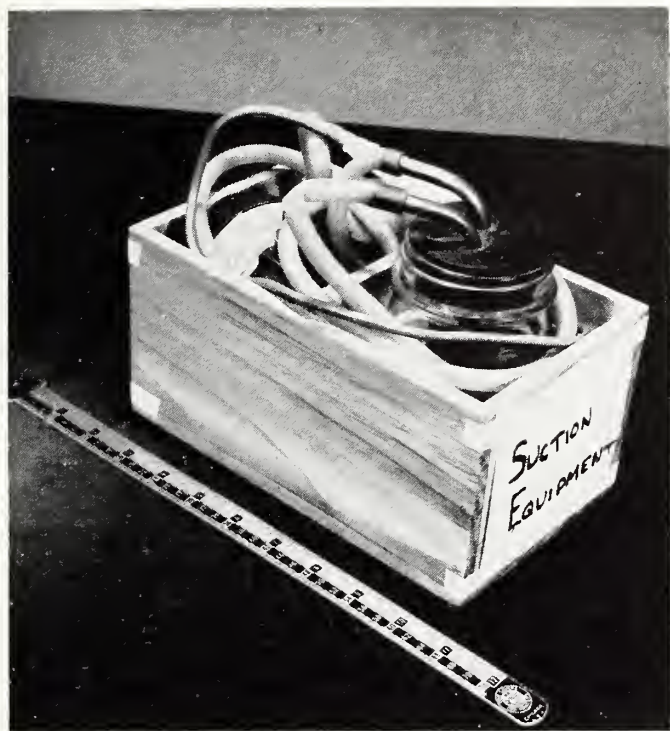
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# An Inexpensive Portable Suction Apparatus

CLEMENT S. DWYER, M.D., PHILIP B. THOMAS, M.D. AND WARREN G. STROUT, M.D.\*



Often, the unconscious, anesthetized or severely ill patient needs oral, nasal, pharyngeal or tracheal suctioning to clear his airway when electrically powered or other expensive mechanical apparatus is not immediately at hand. This need may occur during transportation through corridors, in elevators or even on the ward when there is a lack of machines because of extensive use of the usually available apparatus. Sometimes there may be electrical failure in the line or in a machine.

There are various pieces of apparatus similar to ours on the medical market today but high cost prevents their widespread use at all times and in all places where they might be required. We have found hand bulb manually operated equipment usually inefficient.

Recognizing the need of a number of suction apparatus throughout the hospital, we sought the least expensive and most effective mechanical equipment available. The apparatus pictured above was devised. It consists of a simple  $8\frac{1}{2}'' \times 5'' \times 4''$  divided wooden container with a wide-mouthed pint reservoir jar, a No. 12 two-hole rubber stopper with two bent pieces of copper tubing for inlet and outlet. Suction is provided by a

step-on football pump. This pump is self-inflating by means of a steel coiled spring inside the plastic accordion-like circular sidewalls. There are one-way inlet and outlet valves at its base. A catheter or oropharyngeal metal suction piece is attached by rubber tubing to the inlet copper tube of the reservoir jar. The copper outlet is connected by rubber tubing to the inlet orifice at the base of the pump.\*\* One must be certain that the rubber tubing is attached to the proper nipple.

We have been able to show maximal negative pressure (suction) with this apparatus of about 20mm Hg. It is possible to remove large volumes of gases or liquids into the reservoir by rapid up and down action of the pump. The pump may be stepped on for compression to keep the hands free or pushed with one or both hands with similar ease.

The total cost of this apparatus is under \$3.00.

\*\*The plastic pump was bought from Radio Shack, Boston, Massachusetts.

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# Intermittent Peritoneal Dialysis In Salicylate Intoxication — Report Of A Case

EDWARD J. HUGHES, JR., M.D.\*

The use of the peritoneum as a dialyzing membrane began on an experimental basis in 1923.<sup>1</sup> Because of many complications, it was abandoned in favor of the artificial kidney. The latter, however, has been limited to larger medical centers since it requires trained expert personnel to insure safe operation, which in turn is an extremely costly procedure and not without its hazards even with trained technicians.

With the development of plastic catheters that successfully remove fluid from the peritoneal cavity, and the availability of antibiotics to prevent infection, peritoneal lavage has recently returned as a method of removing toxins from the body.

Largely through the work of Grollman<sup>2,4</sup> and Maxwell<sup>5</sup> the new technique for peritoneal dialysis has become available for the management of several acute emergencies. Its primary use has been with acute renal failure,<sup>5</sup> however, it has also been used successfully in treating hypernatremia,<sup>5</sup> hypercalcemia,<sup>5</sup> salicylate intoxication,<sup>5</sup> barbiturate poisoning<sup>6</sup> and intractable edema.<sup>7</sup> It has also been shown to be of use in some chronic cases such as nephritis with acute episodes.<sup>8</sup>

Because of its simplicity and relative safety, the method utilized below is particularly useful in an area where an artificial kidney is unavailable.

## CASE REPORT

PSM (EMGH 99872). A six-year-old female was admitted to the Eastern Maine General Hospital for the first time on March 5, 1962, in a comatose condition. She had been well up to approximately 5 days previously when she developed a mild fever, headache and general malaise. There was what the mother called "flu" in other members of the family and there was no concern over the child until she seemed to continue with her illness longer than the others. She was treated, according to the mother with occasional aspirin, 5 grain tablet, and liquids and there was no particular change in her condition until the night before admission at which time she seemed to be more feverish and started breathing deeply. During the early morning hours the breathing became even deeper and the child began having hallucinations, became very irrational and irritable. She was seen by a physician in the early morning and immediate hospitalization advised.

Physical examination revealed a 6-year-old well developed, well nourished female appearing acutely ill in a semi-comatose condition, aroused only by deep stimuli which in turn brought on hallucinations and screeching. There were rapid deep respirations and the skin appeared pale and somewhat clammy. The head was normocephalic without masses or tenderness.

The neck was supple. Eyes, ears, nose and throat were unremarkable. The lungs were clear to percussion and auscultation. The heart had a rapid sinus rhythm at 132 per minute. There were no murmurs or thrills and no apparent cardiomegaly. The abdomen was soft without masses or tenderness. Liver, spleen and kidneys could not be palpated. The reflexes were hypo-active but equal bilaterally and there were no obvious pathological reflexes.

The temperature on admission was 101° (F) rectally, the pulse was 132/min., respiration 40/min. and the blood pressure 100/60.

Laboratory work on admission: The hemoglobin was 12.5 gms. 80%, white count -23,100 with 88 polys, 2 bands and 10 lymphocytes. Red cells and platelets appeared normal. CO<sub>2</sub> was 7.5 meq./L. The blood sugar was 115 mgms, potassium 6.4 meq., sodium 135 meq., BUN 28 mgm, and salicylate level -61 mgm. A spinal tap was performed shortly after admission which revealed 142 crenated red cells, no white cells. The spinal fluid contained 17 mgm. of protein and 78 mgm. of sugar.

It was obvious after the laboratory reports that the child had salicylate intoxication with secondary cerebral hemorrhage. After several talks with the mother and other members of the family it was determined that not only was the mother giving this child 10 gr. of aspirin every 3 to 4 hours but a somewhat older sister was playing nurse to this patient and also giving aspirin at frequent intervals, the amount of which was not able to be determined.

Treatment of the salicylate intoxication was started immediately. She was put into oxygen because of deep breathing and a rather grayish color. She was given vitamin C intramuscularly and IV Mephyton® because of the presence of the red cells in the spinal fluid. An intravenous cut-down was started in the left leg and 5% dextrose and saline with added sodium bicarbonate was initiated. After about 6 hours of intravenous therapy a repeat CO<sub>2</sub> was performed and there was no change. Nor was the condition of the child much improved, if at all. It was decided at this point to initiate peritoneal dialysis. This was performed in the method of Maxwell<sup>5</sup> by inserting a plastic catheter through the mid-line of the abdomen into the peritoneal cavity. Then she was flushed out with a peritoneal dialysis fluid over the next 30 hours, during which time 25,000 cc of fluid was dialyzed at 1,000 cc intervals. Each 1,000 cc of fluid was left in the peritoneal cavity approximately 45 minutes and then allowed to drain. This took approximately 15 minutes. Even after 3 hours of lavage the patient's responses improved and after 15 hours of dialysis the CO<sub>2</sub> had risen to 15.5 meq. and the salicylate level had dropped to 28 mgm. A dialysate taken during the night was shown to contain 6.1 mgm. of salicylate per 100 cc. At this time the child was able to sit up and speak intelligently and she started taking fluids well by mouth. The dialysis was continued for approximately 30 hours total, during which time it is estimated at least 50 grains of salicylate was removed.

On the morning following discontinuation of the dialysis, the CO<sub>2</sub> was 24 meq., chlorides 103 mgms, potassium 3.8 meq., sodium 147 meq. and salicylate level 3.8 mgms. At

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this stage the child was perfectly alert, smiling and taking oral fluids well. The urine output was good and intravenous therapy was discontinued. She was watched closely over the next few days but there were no particular unusual signs and only an occasional complaint of a headache. No abnormal neurological signs developed and she was discharged on the 8th hospital day after a completely negative physical examination.

#### DISCUSSION

It is obvious to the writer that the recently revised method of peritoneal dialysis is a safe and effective method for removing toxins from the body. Not only is the apparatus simple and easy to initiate but it is also inexpensive and can be purchased much the same as intravenous fluids and kept in stock for use at any time. Most of the major manufacturers of intravenous fluids have the dialysis solutions and equipment available under various trade-names.

The infusion solutions contain ideal concentrations of electrolytes but devoid of the toxins or electrolytes which are to be removed; such as; salicylate, urea, barbiturate, etc. Two solutions are available. They differ only in their dextrose content — One is 1.5% and the other 7% dextrose. The 1.5% dextrose is most useful in removing abnormal solutes or abnormal amounts of solutes from the general circulation. The other solution containing 7% dextrose is useful in establishing a negative water-balance in edema states. The composition is as follows:

Sodium 140.5 meq/l	Chloride 101 meq.
Calcium 3.5 meq.	Lactate 44.5 meq.
Magnesium 1.5 meq.	Dextrose 1.5% or 7%

Potassium is not present in the commercial preparation because so often it is used to lower a serum potassium. However, if the patient's potassium is determined to be normal, the addition of 4 meq. of potassium chloride per 1000 cc. will prevent hypokalemia.

Briefly the procedure consists of making a small incision in the mid-line of the abdomen below the umbilicus down to the peritoneum, which in turn is pierced by a trocar and the special plastic catheter is inserted through this incision into the peritoneal cavity after suitable local anesthesia. After its insertion the catheter is attached to the sterile plastic tubing draining the infusion bottles. The solution is allowed then to enter the abdominal cavity by gravity feed and is left in the abdominal cavity for approximately 60 minutes, during which time the toxic metabolites diffuse into it through the peritoneal membrane. At the end of this time the diffusion bottles are lowered from the elevated position to the floor and the fluid is drained back into the original bottles by gravity through the closed sterile system and is discarded. This procedure is then repeated for about 24 hours and by the end of the period considerable quantities of the undesirable substances have been removed from the patient.

It is also obvious, since the molecules can pass in both directions that if the patient had an abnormal concentration of any electrolyte this will tend to approach normal values as equilibrium is established between the solutions infused and the body fluids.

It is very important to keep an exact protocol of the patient's fluid balance during this procedure since complications may ensue if most of the fluid is not recovered.

Before the fluid is infused a small amount of aqueous heparin and an antibiotic is instilled into the fluid. The heparin is used to prevent the development of fibrin clots and the antibiotic to prevent any infection.

The amount of solution instilled at each dialysis varies with the size of the patient. New-born infants require only 250 cc of solution and adults require 2000 cc.

The amounts for younger children is easily determined by the amount of abdominal distention noted as the fluid is instilled.

The only contra-indications to the use of peritoneal dialysis are peritoneal infection and recent or extensive abdominal surgery.

After the procedure has been initiated a special nurse can easily perform the infusion and drainage after a few specific instructions. She should obviously not be expected to care for any other patients, but does not require any help.

#### SUMMARY AND CONCLUSIONS

A case of salicylate intoxication requiring rapid therapy is presented. Successful treatment was accomplished by the use of peritoneal lavage. Its advantages over intravenous therapy, exchange transfusion or the artificial kidney are; the rapidity of response, relative ease and safety of performance and low expense of the procedure as is shown by the above report.

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# Routine Or Selective Exploration Of The Post-Partum Uterus

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It has been stated frequently, but still bears repeating, that hemorrhage remains one of the three leading causes of maternal death. (Toxemia-hemorrhage-infection) Birth is a natural function which is associated with loss of a certain amount of blood. Obstetricians have become accustomed, so to speak, to "the sight of blood." In this lies a grave danger. One must not become sophisticated in the attitude towards blood-loss. Each delivery should be conducted with an eye towards preservation of blood.

Prevention of bleeding, at delivery as well as in the post-partum period, has always been a prime objective in Obstetrics. The ways of attaining this goal have varied with the means and methods available. Their effectiveness and inherent dangers have been weighed against the threat of hemorrhage. And so, in this manner, the uterine exploration has been evaluated down through the years.

## HISTORY

It has always been and still is (1962) the formal obstetric teaching that "the uterus should never be invaded if it can be avoided" (Duncan E. Reid, M.D.). The basis for this reluctance to enter the post-partum uterus is, of course, the fear of introducing infection. Valid reasons for this fear could be seen repeatedly in patients on whom a manual removal of the placenta had been performed. H. Halsey<sup>3</sup> commented in 1952 that "its performance is still fraught with many dangers" He found the puerperal morbidity 4 times higher after manual removal of the placenta than in the general clinic population.

And yet, only 2 years later R. L. Hoffman<sup>5</sup> wrote on the "Routine manual removal of the placenta." In a large series of patients he concluded with a total morbidity of 2.25% and a real morbidity of 1.12%. This was considerably less than that quoted by most investigators and also less than that quoted by H. Halsey for the general clinic population. As an explanation he offered the long known close connection between excessive blood-loss and post-partum morbidity. Maybe resistance in the patient following manual removal as practiced earlier had been severely depressed because of excessive blood-loss.

From 1954 until today several articles<sup>1,2,4,7,8</sup> have dealt with the subject of routine post-partum exploration of the uterus. They have all emphasized the dual problem involved; the success in decreasing hemorrhage

versus the risk of infection. Other accomplishments of the routine exploration also have been considered of some importance and will be later described.

## THE TECHNIQUE OF UTERINE EXPLORATION

Reference has been made to the routine manual removal of the placenta. This is not what is meant by the routine post-partum exploration of the uterus. The delivery is carried out in the usual manner, including delivery of the placenta. Immediately following extrusion of the placenta the operator washes his hands in sterile water contained in a water-basin. The patients vulva and perineum are also washed off using wet cotton-balls. Use of a gauze wrap-around or mitt to cover the exploring hand is advocated by most. Without further preparation the gauze-covered hand is introduced into the uterus. The other hand is on the patients abdomen to facilitate moving the uterus, to act as counter-pressure and to aid in the bimanual palpation. It is recommended that a systematic search of the inside of the uterus be made, evaluating in any order or sequence: the anterior wall, the posterior wall, the side-walls and the top. The walls are wiped clean of any placental fragments or pieces of membranes remaining. The normal palpatory findings of the post-partum uterus can only be learned by experience. Usually the walls give a firm, corrugated sensation to the palpating hand. Occasionally a shaggy, irregular and somewhat soft area will be felt. This represents one version of the placental bed. An urge to dig assiduously at this "to remove all tissue," should be suppressed, lest one finds that the intrauterine hand has suddenly become intra-abdominal.

Inspection of the cervix and the vaginal walls for lacerations should of course be done as usual. If any deep cervical laceration is found the exploration affords a good opportunity to ascertain its extent.

## DISCUSSION

Selective exploration of the post-partum uterus has been recommended for years in circumstances such as; following version and extraction, other situations when uterine rupture might be reasonably suspected, incomplete removal of the placenta, excessive post-partum bleeding, etc. This is what may be termed conservative selective exploration. More liberal use of the selective exploration comes in all degrees and gradually approaches the routine usage.

A number of disease-states or conditions, in addition to those mentioned and having been suspected on the basis of clinical information, can be either confirmed or denied by findings at exploration:

1. Congenital malformations of the uterus. (Suspicion aroused from uterine configuration-fetal presentations or other.)
2. Uterine tumors — particularly uterine fibroids.
3. Uterine diverticulae (rare).

The routine exploration has this to offer beyond the selective exploration:

1. Detection of conditions as mentioned, but *unsuspected*.
2. The secure knowledge that the uterus has been emptied.
3. Experience in technique and interpretation.
4. Stimulate uterine contractions. (It has been the experience of many of the writers on the subject that the presence of the intrauterine hand incites the uterus to contract.)

As mentioned before, the dual problem of risks involved versus advantages gained must be the means of evaluation.

Several investigators have testified that the "invasion of the puerperal uterus under antiseptic conditions is a benign procedure" (Lester et. al.)<sup>7</sup>

Series with controls have shown no difference in morbidity between explored and unexplored groups.<sup>4</sup>

The advantages to be gained have already been enumerated. The hope has been primarily in decreasing the incidence of delayed post-partum hemorrhage. The most frequent cause of uterine hemorrhage 24 or more hours post-partum seems to be still a matter of dispute. Some hold that placental fragments rank highest. Others think that subinvolution of the placental site is the more common. F. J. Hofmeister<sup>6</sup> found retained placental fragments as the probable cause in 41%. R. J. Hawkins<sup>4</sup> goes further and states that "in every instance (of post-partum bleeding in patients seen by him) the cause was retained placental tissue" — even though this followed deliveries where the placenta had been inspected and declared to be intact. In a discussion of the paper where this statement appeared M. R. Lazar disagreed stating: "The majority of cases in which the patients return to the hospital in the late puerperium because of bleeding are not specially caused by retained placental tissue. Dieckman substantiates this in his report in which he found that curettage rarely revealed placental tissue. Our experience is essentially the same."

Whatever opinion one may hold it seems difficult to deny that the complete removal of all tissue in the delivery-room would at least diminish the frequency of delayed post-partum hemorrhage.

However, even the routine exploration does not seem to give absolute assurance of an empty uterus. P. D. Mozley<sup>8</sup> reported 9 instances of post-partum bleeding

due to retained tissue in a series of 3022 deliveries which had been followed by exploration. Of these nine, five had been done by physicians inexperienced in the procedure. He states that there seems to be an inverse relationship between remaining tissue after exploration and the experience of the operator.

#### ACCEPTANCE OF THE ROUTINE EXPLORATION

Authors quoted include experienced clinicians and represent a number of recognized institutions.

Approximately 2 years ago, during my residency, I reviewed 1255 charts from the Obstetrical Service of The Toledo Hospital, Toledo, Ohio. The Toledo Hospital is a private institution with several hundred staff-physicians of varied backgrounds and representing a fair cross-section of the country's medical schools. The deliveries had been performed by General Practitioners as well as by specialists certified by The American Board of Obstetrics and Gynecology. No departmental policy on the subject of uterine exploration prevailed. The uterus had been explored following 85% of all deliveries. This figure indicated a high acceptance-rate of the post-partum uterine exploration as a routine procedure for this institution.

#### CONCLUSION

Everyone who does Obstetrics is confronted with the question of post-partum uterine exploration. Should it be done selectively, and if so, what should the indications be? Or should it be a routine, to follow each delivery? An attempt has been made at presenting the arguments. Conclusions must be drawn by the individual accoucheur.

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## State Of Maine

# Department of Health and Welfare

## State Program Of Cancer Control

STANLEY C. BECKERMAN, M.D., *Director*

We have now passed the mid point of the twentieth century and cancer remains medicine's major problem. It is the second leading cause of death nationally, and, similarly, is the second leading cause of death in the State of Maine. Each year in Maine, 1,700 people die of cancer. Our cancer problem is not much different than one would expect to find in any other area of the country, except for the fact, that, broadly speaking, we have a generally lower average income, and a higher proportion of elderly people. The widespread prevalence of the disease and its high mortality, coupled with the aforementioned factors in Maine, produce fear and apathy which compound the problem of cancer control.

### PREVENTION

It is common knowledge that the best way to control a disease is to eradicate it before it starts. Prevention, is therefore a major bulwark of the Cancer Control Program. In this regard, the Division of Cancer Control, with the cooperation of the Department of Education, has instituted an educational program regarding cigarette smoking and its relationship to lung cancer. This program is being aimed at the high school level, since it is felt that it is in this age group that people begin to smoke. Twenty-six of our high schools are involved in this effort, ranging from Presque Isle in the north to Portland in the south, and encompassing a body of almost 12,000 students.

This program is being operated as a controlled study, to determine whether or not, through a planned educational effort, it is possible to alter favorably the attitudes toward cigarette smoking, and ultimately the smoking habits of our teen-age population. This study is now in its second year and has already attracted national attention. To our knowledge, this is the only controlled study on this subject in progress anywhere at this time.

Our program of prevention also includes a Radiation Film Badge Service, which is carried out in cooperation with the Division of Sanitary Engineering. This includes consultation and supervisory services for all types of radiation (x-ray) sources. We also supply laboratory

services for further location of water supplies which may be radioactive. We offer training to individuals handling or operating radiation sources, and we provide consultation services in general industrial hygiene.

### PUBLIC EDUCATION

The field of public education encompasses a wide area and provides many services. An informed populace should be able to avoid carcinogenic agents whenever possible and, therefore, in many instances, the disease may be prevented. By providing films, film strips, speakers, equipment, literature and materials, we continue to broaden our scope in the field of public education. Since the present hope for adequate control of cancer rests with early diagnosis, a well-educated public should, therefore, be aware of the early symptoms of cancer and seek out early diagnosis and treatment.

Since carcinoma of the female cervix should be one of our more easily controlled types of cancer, the Division of Cancer Control has been urging increased activity in the field of Papanicolaou smears (exfoliative cervical cytology). This Division has recently conducted a pilot study in a community general hospital. Under this sponsorship, Papanicolaou smears were offered to all adult females admitted to this particular hospital. Such a program provided multiple services. In addition to providing "Pap" smears for a large number of women who might otherwise not have had such a cancer detection study performed, newspaper publicity of this study provided a broad educational program for the public. Many women were encouraged to seek out similar services in their own private physicians' offices. Private physicians, were, therefore, stimulated to increase performance of "Pap" smears in their own private offices. It is hoped that such programs as this can be instituted in various areas of the state with the same beneficial results.

### PROFESSIONAL EDUCATION

The program of public education accomplishes little or nothing if the physicians of our state are not well-

informed and up-to-date. Therefore, it becomes mandatory to have a constantly aggressive and stimulating program of professional education. This program includes the use of films, mailing pieces, literature and other such materials. The Division of Cancer Control encourages and, when necessary, subsidizes programs whereby investigators working at large cancer centers are brought into various areas of the state for purposes of lectures, clinics and other modes of professional education. This affords the physicians of the area an opportunity to have their individual questions answered and to be brought up-to-date as to what is new in the field of cancer. Such a program also provides free consultation service with the particular visiting physician. In this way, utilizing programs which offer multiplicity of services, we hope to improve the care of cancer patients throughout the State. Similar educational programs are designed and aimed at the organized professions of nursing, dentistry, pharmacy, as well as other interested groups.

#### CASE FINDING

There are now eight tumor clinics operating within the State of Maine. The Division of Cancer Control supports these clinics with material services, loaned equipment, financial participation and the purchase of services. Most of these tumor clinics act as centers for the diagnosis and treatment of malignant disease. The State-subsidized clinics deal with about 2,200 patients per year, making 3,500 patient visits annually. Of these, almost 500 patients were diagnosed as having malignant diseases. In connection with these clinics, the Division of Cancer Control provides training for hospital, clinic or laboratory staff.

#### RESEARCH

The control of cancer and its ultimate cure will be an outgrowth of present day research. Here in the State of Maine, cancer research is being conducted at the Jackson Memorial Laboratories in Bar Harbor, mainly in the fields of basic sciences, at the University of Maine from a biochemical point of view, and at the Thayer Hospital in Waterville from the point of view of clinical chemotherapy. The Division of Cancer Control has a continuing program to stimulate and assist cancer research whenever and wherever possible.

#### COORDINATION AND COOPERATION

In order for a Cancer Control Program to be meaningful and adequate, the needs of the population must be clearly determined and known before such a program can be put into operation. In order to determine such needs, the Department's Division of Research and Vital Records cooperates with the Division of Cancer Control in order to present the picture of cancer in the State of Maine. To further determine and fulfill the needs of the people of Maine, the Division of Cancer Control cooperates with the organized medical, dental, nursing and pharmaceutical professions within the State, as well as the various voluntary health agencies. A Cancer Committee has been appointed by the Maine Medical Association, which acts in an advisory capacity to the Division of Cancer Control in order to help clarify the needs of the population of Maine. The Division of Cancer Control has a continuing program of cooperation and coordination with the United States Public Health Service and the National Cancer Institute in implementing the Cancer Control Program in our State.

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#### Perils Of Mail Order Pharmacies

Doctors should urge their patients to think twice before succumbing to the temptations of mail-order economy in the area of ethical pharmaceuticals. The delays inherent in mail-ordering as well as the uncertainty as to what is delivered — and when — should convince the reasonable patient that he is ahead by having his prescription filled by his regular local druggist. — Editorial in *Wisconsin Medical Journal*, March 1962.

## Maine Heart Association Notes



### The Management And Control Of Rheumatic Fever

"The most recent chapter in the long story of the relationship of the Group A streptococcus to rheumatic fever has been the demonstration that *initial* attacks of rheumatic fever in the general population may be prevented by prompt and adequate penicillin therapy of the antecedent infection. Certain chemotherapeutic principles upon which this preventive approach is based have been firmly established as a result of the admirably controlled large-scale studies conducted at the Streptococcal Disease Laboratory at Fort Francis E. Warren Air Force Base. These principles may be summarized as follows.


To prevent the complication of subsequent rheumatic fever, Group A streptococci must be eradicated completely from the nose and throat of the patient with streptococcal pharyngitis. Clinical cure of the signs and symptoms of the infection is inadequate if organisms persist after treatment. Sulfonamides, which are bacteriostatic, do not eradicate the streptococcus. Penicillin, although a powerful bactericidal agent, must be administered well beyond the period of clinical cure to eradicate streptococci from the throat.

The therapeutic regimen required to achieve this bacteriologic cure has been clearly defined. If penicillin is administered orally, a minimum daily dose of 500,000 units must be given for at least 10 days. Larger doses given for shorter periods are less effective. If penicillin is administered parenterally any schedule may be employed which maintains effective blood concentrations of penicillin for a 10-day period. Group A streptococci are invariably sensitive to penicillin levels of less than 0.04 units per milliliter *in vitro*. The blood concentration need not be high, therefore, to achieve a maximal killing effect *in vivo*. Once these low blood levels have been achieved, the efficiency of chemotherapy is a function of the duration for which they are maintained.

. . . . Therefore a single injection of benzathine penicillin, particularly in the larger dose of 1.2 million units, will achieve a cure of streptococcal pharyngitis, both clinically and bacteriologically, as efficiently as any other course of penicillin administered in any dose for 10 days.

It is apparent that the major limitation of the chemotherapeutic approach to the prevention of initial attacks of rheumatic fever is the difficulty of clinical identification of streptococcal sore throat and the fact that at least one-third of patients with acute rheumatic fever do not appear to have had a clinically apparent infection that would have been brought to the attention of the clinician for treatment. . . . The syndrome of sudden onset of fever, sore throat, 'beefy' redness and pharyngeal exudate suggest the diagnosis with about 70 per cent certainty. In infants and very young children, however, the difficulties of diagnosis are far greater because these symptoms and signs are less apparent. Furthermore, very mild-looking signs may be due to streptococcal infection, and conversely, some viral infections produce exudates and signs identical with those of streptococcal pharyngitis.

For this reason, the throat culture is an invaluable guide to the indications for therapy. Waiting for 24 hours before instituting therapy, and until a preliminary report is made on the presence or absence of hemolytic colonies on a sheep's blood agar plate inoculated with a throat swab, will not jeopardize the patient's recovery or the prevention of rheumatic fever. Indeed it is only by the use of throat cultures that the treatment of upper respiratory infections can be placed upon a sound scientific basis at the present time. It is well to remember that more than 90 per cent of upper respiratory infections are due to viral rather than bacterial causes."



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1. Best, C. H., and Taylor, N. B.: *The Physiological Basis of Medical Practice*, ed. 6, Baltimore, The Williams & Wilkins Company, 1955, p. 578.

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## County Society Notes

## YORK

October 10, 1962

A meeting of the York County Medical Society was held at the York Hospital in York, Maine on October 10, 1962. James S. Johnston, M.D. presided in the absence of the President.

A lively discussion of after care of surgical cases was discussed by several members. Walter R. Peterlein, Jr., M.D. was elected to represent the society as a member of the Maine Medical Association's sub-committee on recruitment. The society went on record as opposed to any change in the Maine Medical Association Council set-up. It was voted to accept suggestions made by Francis S. Sleeper, M.D., Superintendent of the Augusta State Hospital, relative to State Hospital reports.

John J. Lorentz, M.D., Medical Director of the Hyde Memorial Rehabilitation Center in Bath, Maine, gave a very interesting talk on the work at the Center which was followed by a moving picture of the after care of cases of cerebral hemorrhage.

CHARLES W. KINGHORN, M.D.  
*Secretary*

## LINCOLN-SAGADAHOC

October 16, 1962

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at the Ledges in Wiscasset, Maine on October 16, 1962.

Harry M. Wilson, M.D. was appointed to represent the county society as a member of the Maine Medical Association's subcommittee on recruitment.

Francis A. Winchenbach, M.D., Chairman of the Maine Medical Association's Health Insurance Committee, discussed the plans of the Associated Hospital Service of Maine for those over 65 years of age.

Ralph C. Stuart, M.D., President of the Maine Medical Association, spoke on the Association's Student Loan Fund and on the public image of the medical profession. He discussed also the proposed changes in apportionment of the Council and of the Executive Committee.

Merrill J. King, Jr., M.D. of Rockland exposed the mysteries of tonometry and spoke briefly on the etiology, physiology and treatment of glaucoma.

GEORGE W. BOSTWICK, M.D.  
*Secretary*

## PENOBSCOT

October 16, 1962

A meeting of the Penobscot County Medical Society was held on October 16, 1962 at the Oronoka Hotel in Orono, Maine. Forty-eight members and guests were present with the President, Clement A. Dwyer, M.D. presiding.

The speaker of the evening, Warren Wacker, M.D. of Harvard Medical School, gave an illustrated talk on the urinary excretion of the enzyme lactic dehydrogenase and the use of this test in diagnosis of renal disease. The lecture represented original studies and findings by Dr. Wacker.

Drs. Thornton W. Merriam, Jr. and Anders T. Netland, both of Bangor, were elected to membership in the society.

The report of the Polio Committee was given showing that the Polio Pow Wow clinics held on October 14, to provide type II oral vaccine to the people of Penobscot County, ad-

ministered a total of 38,907 doses. The total of each clinic site is as follows: Patten-479, Millinocket-7900, Lincoln-3630, Howland-1683, Orono-3283, Old Town-5579, Dexter-1755, Charleston-1180, Brewer-5983, Bangor-7435. This total added to that given in the Bangor schools (7373) and Dow Air Force Base (5800) in the week preceding the clinics, makes a total of 52,080 doses given.

H. Draper Warren, M.D. was elected to represent the county society as a member of the Maine Medical Association's sub-committee on recruitment.

The report on the coming TV series was read. These are the "Meet your Doctor" programs arranged by the Public Relations Committee and are to be on WABI-TV as follows:

January — Radiation; February — Rheumatic Fever; March — Urological Diseases; April — Care of the Indigent; May — The Hospital Speaks.

Drs. Richard C. Wadsworth, Albert C. Todd and John E. Whitworth were appointed by the President as a nominating committee to draw up a slate of 1963 officers.

It was voted that the Penobscot County Medical Society go on record as being strongly opposed to any legislation permitting chiropractors to treat industrial accident and workmen's compensation cases.

A report of the Emergency Call system for Bangor and Brewer was given by William A. Purinton, M.D. The society voted to accept the recommendations of the executive council in the matter of running the system.

November 20, 1962

The monthly meeting of the Penobscot County Medical Society was held on November 20, 1962. Forty members gathered at the Pilot's Grill in Bangor, Maine and the meeting was conducted by the President, Clement A. Dwyer, M.D.

The speaker of the evening was Dr. A. J. Mackay, Professor and Chairman of the Department of Surgery, University of Vermont Medical School. Dr. Mackay discussed the development of the surgical microscope and its use in microsurgery. He presented animal studies and some clinical reports concerning the use of this technique in surgery of the Vas Deferens, bile ducts, blood vessels, ureters and lymphatics. Colored slides were shown.

Charles D. McEvoy, Jr. M.D. gave a report on the last meeting of the House of Delegates of the Maine Medical Association discussing the highlights of the session.

The Bidwell committee report, which had been sent to all county society members, was discussed. The majority opinion of the members was that reorganization of the governing body of the State Society as outlined by the report would not improve the present status because it would: 1) provide an unwieldy group of councilors. 2) produce an actual governing body that was too small. 3) give certain counties or areas more governing influence than they should have. It was voted that the county society go on record as being opposed to the content of this report.

The following officers were elected for 1963:

President, Allison K. Hill, M.D., Bangor  
 President-elect, William A. Purinton, M.D., Bangor  
 Treasurer, Benjamin L. Shapero, M.D., Bangor  
 Secretary, Frederick C. Emery, M.D., Bangor  
 Councilor, Herbert C. Gilman, M.D., Millinocket  
 FREDERICK C. EMERY, M.D.  
*Secretary*

#### CUMBERLAND

October 18, 1962

Sixty members and guests were present at the meeting of the Cumberland County Medical Society which was held at the Eastland Motor Hotel in Portland, Maine. The meeting

was called to order by the President, Robinson L. Bidwell, M.D.

Drs. Alice N. Cunningham and Rudolf G. Winkelbauer, both of Brunswick, were elected to membership.

A motion was made by Edward G. Asherman, M.D., that the delegates be instructed to vote in favor of the changes in the Council of the Maine Medical Association as read. This was seconded. An amendment was made by Alvin A. Morrison, M.D. that there be made after Page II, Sentence II in reference to the terms of service on the committee "No member is to serve for two consecutive terms," and that the delegates be instructed to press for this change. The amendment was seconded and passed. Benjamin Zolov, M.D. asked for clarification of the duties of the proposed executive committee. In answer, Dr. Bidwell explained that he feels that the sphere of action of the Council was too broad and its duties too many. It was proposed that the Council retain those duties relating to financial matters, public relations, and ethics, and that the executive committee would undertake all other duties now performed by the Council.

Ralph C. Stuart, M.D., President of the Maine Medical Association was introduced by Dr. Bidwell and asked to comment. He reported that the committee report under discussion has been rejected by all the county societies whose meetings he has attended to date. Following further pro and con discussion, a vote was called for, and Dr. Asherman's motion, amended by Dr. Morrison, was passed almost unanimously. A few further remarks were made by Dr. Stuart who stated that he now understood the reasons for the committee recommendations and felt that Cumberland County was sincere in its efforts to improve medical organization in the State of Maine. He further remarked that he would communicate this information to the other county societies.

November 15, 1962

A meeting of the Cumberland County Medical Society was held at the Eastland Motor Hotel in Portland, Maine on November 15, 1962. After a social hour and dinner the meeting was called to order by the President, Robinson L. Bidwell, M.D.

Michael D. Ballard, M.D. of Portland was elected to membership in the society. A resolution by Albert C. Johnson, M.D. on the death of Henry P. Johnson, M.D. was read and it was moved that this be spread on the minutes of the society and a copy be sent to his wife.

The President announced that the December meeting would be the annual meeting to include the election of officers. Drs. Warren C. Baldwin, Burton L. Olmsted and John F. Gibbons were appointed by the President as a nominating committee to bring a slate of officers including the President, Vice-President, delegates and alternates to the Maine Medical Association and three members of the Public Relations and Grievance Committee.

Dr. Bidwell commented on the excellent job done by Clement A. Hiebert, M.D. and his committee in organizing and running the Fall Clinical Session of the Maine Medical Association which was held in Portland, Maine in October. He further stated that Mr. Paul A. MacDonald, Secretary of State and Registrar of Motor Vehicles, wished to bring a bill in the Legislature requiring physicians to report mental and physical defects of patients which would make them dangerous drivers of motor vehicles. Mr. MacDonald felt that the bill would not pass unless approved by a medical group and desired the approval of the medical society. Dr. Bidwell asked for a motion to implement this request. Several additions were made to provide such a motion, but failed passage. There was much discussion by many members of the society, pro and con, although there appeared to be general agreement that passage of such a statute would be a desirable thing. A motion was made by John B. Titherington, M.D. that the chair appoint

a committee to study the problem and to bring back recommendations as to whether the society approve such a law. This was seconded and passed and the following committee was appointed: Maurice Van Lonkhuyzen, M.D., Chairman, Daniel P. Storer, M.D. and John B. Titherington, M.D.

Final item on the agenda was a talk by Mr. Robert Pacios, CPA, of Lewiston on "Practical Aspects of Income Tax Returns" which was followed by a question and answer period.

ALBERT ARANSON, M.D.  
*Secretary*

## KENNEBEC

October 18, 1962

A meeting of the Kennebec County Medical Association was held at the Jefferson Hotel in Waterville, Maine on October 18, 1962. Brinton T. Darlington, M.D. called the meeting to order in the absence of the President.

The following physicians were elected to membership: Drs. Richard T. Chamberlin and Jose Rodriguez, both of Waterville, and John H. Shaw of Augusta by transfer from the Cumberland County Medical Society.

Paul H. Pfeiffer, M.D. spoke on the recruitment program for medical students and asked for the opinion of the Association concerning a direct grant program to needy medical students and without specific requirements for practicing within the State of Maine, saying that he wanted to have the Maine Medical Association request the State Legislature to release state funds for such grants. Unanimous consent was given by the Association.

Morton A. Madoff, M.D., assistant physician with the infectious disease unit at the New England Medical Center and a senior instructor in medicine at Tufts University College of Medicine, was the guest speaker. He spoke on the viral vaccination and immunization programs especially in regard to poliomyelitis and influenza. He stated that he could not agree with the United States Public Health Service in allowing children to take the type III Sabin vaccine. He said that he feels that it is possible for children to pass the virus on to adults within the family group and probably as a safety precaution the type III should be withdrawn until further studies have been made. He also disagreed with the Public Health Service's suggestion of mass influenza vaccination saying that the length of immunity was unknown and that immunizations were truly indicated in pregnant women, people with chronic heart and renal disease and in people over 65 years of age. Many questions were raised from the floor and most interesting discussions followed.

November 15, 1962

The Kennebec County Medical Association held its month-

ly meeting at the Veterans Administration Center in Togus, Maine on November 15, 1962. George J. Robertson, M.D. called the meeting to order.

Paul H. Pfeiffer, M.D. reported on the "Bidwell Report" which was followed by discussion. A nominating committee was appointed to bring in a slate of officers for the coming year.

Joseph S. Weltman, M.D., director of the Veterans Administration Center, welcomed the Association to the Center and Robert L. Ohler, M.D., chief of the medical services, introduced the speaker of the evening. Clement A. Hiebert, M.D. of Portland, clinical associate at the Massachusetts General Hospital and assistant in surgery at the Harvard Medical School, spoke on "Pulmonary Atelectasis — A Method of Prevention and Treatment Without Drugs or Instruments." He was assisted by Miss Marica Wasenius, Head of the Chest Physiotherapy Unit at the Massachusetts General Hospital. Miss Wasenius demonstrated the instructions and exercises given to patients pre-operatively to prevent post-operative pulmonary complications and then demonstrated the physiotherapeutic measures taken to overcome atelectasis.

EARLE M. DAVIS, M.D.  
*Secretary*

## New Members

### CUMBERLAND

Robert A. Bearor, M.D., Maine Medical Center, Portland  
Alice N. Cunningham, M.D., 32 Federal Street, Brunswick  
David Naide, M.D., 500 Forest Avenue, Portland  
Norman W. Saunders, M.D., 12 Deering Street, Portland  
Rudolf G. Winkelbauer, M.D., Baribeau Drive, Brunswick

### KENNEBEC

Richard T. Chamberlin, M.D., 14 Gilman Street, Waterville  
Jose Rodriguez, M.D., 14 Gilman Street, Waterville

### LINCOLN-SAGADAHOC

Charles E. Burden, M.D., 1 North Street, Bath  
Alexander G. Stetkevych, M.D., 858 Washington Street, Bath

### OXFORD

Ottone Renzulli, M.D., 426 Franklin Street, Rumford  
Charles M. Smith, M.D., Dixfield

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Volume 53, Number 6  
June, 1962

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763 Congress St., Portland  
18 Bramhall St., Portland  
Ward Town Rd., Freeport  
188 State St., Portland  
323 Main St., Cumberland Mills  
55 Stroudwater St., Westbrook  
52 Belmont St., Portland  
312 Congress St., Portland  
22 Bramhall St., Portland  
73 Deering St., Portland  
235 State St., Portland  
173 Pleasant Ave., Portland  
83 West St., Portland  
107 Main St., Yarmouth  
22 MacMillan Dr., Brunswick  
Flaggy Meadow Rd., Gorham  
690 Congress St., Portland  
690 Congress St., Portland  
690 Congress St., Portland  
73 Deering St., Portland  
22 Bramhall St., Portland  
58 Deering St., Portland  
9 Deering St., Portland  
Box C, Pownal  
38 Deering St., Portland  
12 Pine St., Portland  
Box C, Pownal  
72 West St., Portland  
58 Federal St., Brunswick  
Cumberland Ctr.  
47 Deering St., Portland  
326 Stevens Ave., Portland  
173 State St., Portland  
12 Deering St., Portland  
18 Bramhall St., Portland  
855 Sawyer St., So. Portland  
331 Cottage Rd., So. Portland  
27 Deering St., Portland  
R.F.D. No. 1, West Bridgton  
988 Sawyer St., So. Portland  
131 Chadwick St., Portland  
295 Brighton Ave., Portland  
131 Chadwick St., Portland  
18 Deering St., Portland  
42 Deering St., Portland  
49 Deering St., Portland  
131 Chadwick St., Portland  
171 State St., Portland  
201 State St., Portland  
144 State St., Portland  
192 State St., Portland  
175 Vaughan St., Portland  
310 Main St., Westbrook  
22 Bramhall St., Portland  
131 State St., Portland

#### SENIOR MEMBER

Morrison, James B. Main St., Ashland

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Osborne, John R. Veterans Adm., Togus  
Savage, Richard L. 4 Elm St., Fort Kent

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Herson, Joseph H. 334 E. 25th St., New York 10, N. Y.

### CUMBERLAND COUNTY

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*Secretary-Treasurer* — Albert Aranson, M.D.

#### ACTIVE MEMBERS

Agan, Robert W. 144 State St., Portland  
Allen, Donald E. Sebago Lake  
Analis, Harry 902 Brighton Ave., Portland  
Andrews, Anneliese M. Maine Medical Ctr., Portland  
Ansell, Harvey B. 39 Deering St., Portland  
Applin, Hilton H. 6 Cumberland St., Brunswick  
Aranson, Albert 39 Deering St., Portland  
Asali, Louis A. 29 Deering St., Portland  
Asherman, Edward G. 131 Chadwick St., Portland  
Bacastow, Merle S. 22 Bramhall St., Portland  
Bachrach, Louis 16 Union St., Brunswick  
Baldini, Elio 22 Bramhall St., Portland  
Baldwin, Warren C. 42 Deering St., Portland  
Barnes, Kirk K. 11 McKen St., Brunswick  
Bennet, Eben T. 49 Deering St., Portland  
Bettle, Ronald A. 32 Federal St., Brunswick  
Bidwell, Robinson L. 31 Bramhall St., Portland  
Bischoffberger, John M. Naples  
Bisgrove, John G. 165 Park Row, Brunswick  
Bishop, Lloyd W. 211 Vaughan St., Portland  
Blaisdell, Elton R. 12 Deering St., Portland  
Blumberg, Edward Box C, Pownal  
Bonney, James H. 229 Vaughan St., Portland  
Bove, Louis G. 12 Deering St., Portland  
Bowman, Peter W. Box C, Pownal  
Branson, Sidney R. 37 Main St., So. Windham  
Broggi, Frank S. 18 Neal St., Portland  
Brown, Douglas H. 548 Shore Rd., Cape Elizabeth  
Burnett, Claude A., Jr. 59 Deering St., Portland  
Burns, Robert M. 582 Main St., Westbrook  
Burrage, William C. 57 Deering St., Portland



## HANCOCK COUNTY

President — James H. Crowe, M.D.

Secretary-Treasurer — Russell G. Williamson, M.D.

### ACTIVE MEMBERS

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Coffin, Ernest L. Northeast Harbor  
Coffin, Silas A. 39 High St., Bar Harbor  
Cooper, Llewellyn W. 194 Main St., Bar Harbor  
Crowe, James H. 121 Main St., Ellsworth  
Dolan, Thomas F., Jr. 50 Union St., Ellsworth  
Gray, Philip L. Blue Hill  
Herbert, Walter W. Eastern Mem. Hosp., Ellsworth  
Hsu, Theodore S. 14 High St., Ellsworth  
Joost, Arthur M., Jr. P.O. Box B, Bucksport  
Knickerbocker, Charles H. 15 High St., Bar Harbor  
Kopfmann, Harry Deer Isle  
Lane, Russell M. Water St., Blue Hill  
Larrabee, Charles F. 48 Mt. Desert St., Bar Harbor  
McIntyre, John D. 50 Union St., Ellsworth  
O'Meara, Edward S. Eastern Mem. Hosp., Ellsworth  
Russell, Robert F. Penobscot  
Suyama, Eji 58 W. Main St., Ellsworth  
Thegen, W. Edward Elm St., Bucksport  
Torrey, Marcus A. 75 State St., Ellsworth  
Weymouth, Raymond E. 194 Main St., Bar Harbor  
Wilbur, Herbert T., Jr. P.O. Box 175, Southwest Harbor  
Williams, Thomas W. 50 Union St., Ellsworth  
Williamson, Elizabeth E. Blue Hill  
Williamson, Russell G. Blue Hill Mem. Hosp., Blue Hill

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Babcock, Harold S. Castine

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Brunswick  
Jennings, Richard K. Univ. of Mass., Amherst, Mass.

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Secretary-Treasurer — Earle M. Davis, M.D.

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Ashley, Alta Dist. III Health Office, Augusta  
Barnard, John M. H. 21 Western Ave., Augusta  
Barron, Richard E. Main St., Monmouth  
Bauman, Clair S. 159 Silver St., Waterville  
Beckerman, Stanley C. 82 Elm St., Waterville  
Bolduc, Jean L. 173 Main St., Waterville  
Bourassa, Harvey J. 15 Silver St., Waterville  
Brann, Henry A. 31 Western Ave., Augusta  
Breard, J. Alfred 15 Summer St., Waterville  
Bull, Frank B. 72 Church St., Gardiner  
Canal, Ory D. Augusta State Hosp., Augusta  
Castellanos, Jose Augusta State Hosp., Augusta  
Chasse, Richard L. 18 Park St., Waterville  
Chen, Jen-Ti Cherry Hill Terrace, Waterville  
Cook, Aaron 23 High St., Waterville  
Crawford, Joseph R. 105 Water St., Augusta  
Dachslager, Philip 21 Western Ave., Augusta  
Darlington, Brinton T. Westwood Rd., Augusta  
Davis, Earle M. 2 School St., Waterville  
Denison, John D. 105 Brunswick Ave., Gardiner  
Dennis, Richard H. 33 College Ave., Waterville  
Dore, Clarence E. 2 School St., Waterville  
Dunn, Robert H. Veterans Adm., Togus  
Emanuel, Meyer Veterans Adm., Togus

English, Lena M. Veterans Adm., Togus  
Ervin, Edmund N. 2 School St., Waterville  
Fisher, Dean H. State House, Augusta  
Fisher, Samson 173 Main St., Waterville  
Giddings, Lane 6 E. Chestnut St., Augusta  
Giddings, Paul D. 31 Western Ave., Augusta  
Giesen, Joseph H. 34 Gilman St., Waterville  
Gingras, Adolphe J. 99 Water St., Augusta  
Gingras, Napoleon J. 6 E. Chestnut St., Augusta  
Goodof, Irving I. Thayer Hospital, Waterville  
Goodrich, Blynn O. 165 Main St., Waterville  
Gould, George I. 79 Main St., Richmond  
Guillemette, Maurice R. 109 Water St., Augusta  
Guite, L. Armand 45 Elm St., Waterville  
Harlow, Edwin W. 177 Main St., Waterville  
Herring, Leon D. Memorial Drive, Winthrop  
Hill, Howard F. 33 College Ave., Waterville  
Hill, Kevin 33 College Ave., Waterville  
Hirschberger, Celia 44 Main St., Waterville  
Hornberger, H. Richard 2 School St., Waterville  
Hurd, Allan C. 72 Church St., Gardiner  
Jackler, Jacob M. 14 Gilman St., Waterville  
Jones, Paul A. Jr. 2 School St., Waterville  
Landwehr, George R. 111 Water St., Augusta  
Langer, Ella State House, Augusta  
Lansing, Peter F. 16 Macomber Ave., Augusta  
Lepore, Anthony E. 72 Church St., Gardiner  
Marshall, Joseph A. 177 Main St., Waterville  
Mathews, Hugh J., Jr. 345 Water St., Gardiner  
McLaughlin, Clarence R. 345 Water St., Gardiner  
McLaughlin, Ivan E. 345 Water St., Gardiner  
McQuillan, Arthur H. 177 Main St., Waterville  
Melendy, Oakley A. Doctors Park, 89 Hospital St., Augusta  
Milliken, Howard H. 105 Second St., Hallowell  
Monsivais, Alfredo Augusta State Hosp., Augusta  
Moore, Valentine J. Thayer Hospital, Waterville  
Morris, Craig W. 50 Bangor St., Augusta  
O'Connor, Francis J. 4 Woodlawn St., Augusta  
Ohler, Robert L. Veterans Adm., Togus  
Papadopoulos, George Provincial Mental Hosp., Essondale,  
B. C., Canada  
Peddle, Harry M. K. 23 Western Ave., Augusta  
Pfeiffer, Paul H. 14 Gilman St., Waterville  
Plimpton, Jay R. 283 Water St., Augusta  
Pomerleau, Ovid F. 179 Main St., Waterville  
Pomerleau, Rodolphe J. F. 27 Main St., Waterville  
Poulin, Albert A. Cherry Hill Dr., Waterville  
Poulin, James E. 177 Main St., Waterville  
Pratt, Loring W. 177 Main St., Waterville  
Provost, Helen C. 48 Green St., Augusta  
Provost, Pierre E. 48 Green St., Augusta  
Reynolds, John F. 216 Main St., Waterville  
Richards, Lee W., Jr. 21 Western Ave., Augusta  
Robertson, George J. 33 College Ave., Waterville  
Runyon, William N. 283 Water St., Augusta  
Sanders, Stephen W. 120 Main St., Winthrop  
Saunders, Allen I. Ferry Rd., R.F.D. 2, Augusta  
Schmidt, Lorrimer M. Veterans Adm., Togus  
Schumacher William E. 14 Westwood Rd., MD "B", Augusta  
Schwarz, Harald J. Sisters Hospital, Waterville  
Seligman, Morris J. Veterans Adm., Togus  
Senenky, Joseph P. Augusta State Hosp., Augusta  
Sewall, Kenneth W. 2 School St., Waterville  
Shelton, M. Tieche 61 Winthrop St., Augusta  
Shippee, James N. 122 Main St., Winthrop  
Simpson, Margaret R. Box 275, Togus  
Sleeper, Francis H. Augusta State Hosp., Augusta  
Smith, Kenneth E. Veterans Adm., Togus  
Sommerfeld, Kurt A. 5 Brunswick Ave., Gardiner  
Southern, Edward M. 34 Gilman St., Waterville  
Spellman, Francis A. Veterans Adm., Togus  
Stinchfield, Allan J. P.O. Box 343, Augusta  
Stocks, Joseph F. 67 Silver St., Waterville  
Sturtevant, Vaughn R. 33 College Ave., Waterville  
Towne, Charles E. 18 Common St., Waterville  
Veilleux, Lucien F. 173 Main St., Waterville  
Weltman, Joseph S. Veterans Adm., Togus  
Willard, Harold N. Thayer Hospital, Waterville  
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Kagan, Samuel H. 283 Water St., Augusta  
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Priest, Maurice A. 108 S. Stone St., Deland, Florida  
Risley, Edward H. P.O. Box 143, Prides Crossing, Mass.  
Shannon, Charles E. G. 9 Park St., Waterville

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Reynolds, Ralph L. 216 Main St., Waterville

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## KNOX COUNTY

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Brouwer, Johan 5 Beech St., Rockland  
Dennison, Frederick C. 183 Main St., Thomaston  
Earle, Ralph P. Vinalhaven  
Eddy, Robert H. 5 Beech St., Rockland  
Fuller, Barbara L. 20 Chestnut St., Rockland  
Hawkins, Donald B. Atlantic Ave. & Sea St., Camden  
Heath, Parker, Jr. 22 White St., Rockland  
Hochschild, Hugo 33 Main St., Thomaston  
Hopping, John S. R.F.D. No. 2, Union  
Hunter, Albert L. Knox County Gen. Hosp., Rockland  
Jameson, C. Harold Medical Arts Bldg., Rockland  
Jones, Paul A., Sr. Union  
Kibbe, Frank W. R.F.D., Lincolnville  
King, Merrill J. 22 White St., Rockland  
King, Merrill J., Jr. 22 White St., Rockland  
Lawry, Oram R., Jr. 96 Limerock St., Rockland  
McLellan, William A. 87 Chestnut St., Camden  
Millington, Paul A. 44 Mountain St., Camden  
Morse, Edward K. 22 White St., Rockland  
Onat, Mustafa V. St. George  
Root, John A. 22 White St., Rockland  
Soule, Gilmore W. 22 White St., Rockland  
Tounge, Harry G., Jr. 12 Union St., Camden  
Ward, William W. 76 Limerock St., Rockland  
Wasgatt, Wesley N. 41 Talbot Ave., Rockland  
Waterman, Richard Main St., Waldoboro  
White, Henry O. 22 White St., Rockland  
Worthing, Verla E. Box A, Thomaston

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Campbell, Fred G. P.O. Box 484, Warren  
Hall, Walter D. 407 Main St., Rockland

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Frost, Harold M. Friendship  
Loewenstein, George Chebeague Island  
Winter address — Aripeka, Florida  
Platt, Anna Beauchamp Rd., Rockport  
Winter — 110 Manatee Rd., Belleair, Clearwater, Fla.

## AFFILIATE MEMBER

Waterman, Dorothy Waldoboro

## LINCOLN-SAGADAHOC COUNTY

*President* — Hamdi Akar, M.D.  
*Secretary-Treasurer* — George W. Bostwick, M.D.

## ACTIVE MEMBERS

Akar, Hamdi 17 Grove St., Bath  
Andrews, John F. 20 West St., Boothbay Harbor  
Belknap, Samuel L. Damariscotta  
Betts, Anthony 39 Harpswell St., Brunswick  
Bostwick, George W. Newcastle  
Clark, Richard I. P.O. Box 127, Freeport  
Dalrymple, Sidney C. So. Great Rd., So. Lincoln, Mass.  
Doble, Miriam 990 Washington St., Bath  
Dougherty, John F. 112 Front St., Bath  
Gregory, Philip O. St. Andrews Hosp., Boothbay Harbor  
Griffin, Carl R., Jr. 69 Townsend Ave, Boothbay Harbor  
Hamilton, Virginia C. 900 Washington St., Bath  
Kinder, Edward L., Jr. 1027 Washington St., Bath  
Lenfest, Stanley R. Waldoboro  
Nichols, Arthur A. Edgcomb  
Oceretko, Arkadij 37 Court St., Bath  
Powell, Ralph C. Damariscotta  
Proctor, Thomas E. Boothbay Harbor  
Smith, Jacob 118 Front St., Bath  
Smith, Joseph I. 118 Front St., Bath  
Tracy, Mary J. Bristol Rd., Damariscotta  
Wilson, Harry M. 944 Middle St., Bath  
Winchenbach, Francis A. 910 Washington St., Bath  
Zeller, Alan W. 35 Main St., Damariscotta

## HONORARY MEMBERS

Barrows, Harris C. 5 Oak St., Boothbay Harbor  
Desjardins, Arthur U. South Bristol  
Kershner, Warren E. 57 Green St., Bath  
Morin, Harry F. 905 Middle St., Bath  
Stetson, Rufus E. Damariscotta

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*President* — H. Richard Bean, M.D.  
*Secretary-Treasurer* — Albert P. Royal, Jr., M.D.

## ACTIVE MEMBERS

Akerberg, Ake 10 Maple St., So. Paris  
Aucoin, Peter B. 87 Congress St., Rumford  
Bean, H. Richard 171 Main St., Norway  
Defoe, Garfield G. Dixfield  
Dixon, Walter G. 16 Deering St., Norway  
Elsmore, Dexter E. 11 Main St., Dixfield  
Harper, Harry L. 17 Main St., So. Paris  
Hiebert, Joelle, C., Jr. Box 148, Norway  
Howard, Henry M. 105 Franklin St., Rumford  
Jackson, Norman M. 89 Congress St., Rumford  
Kudisch, Leonidas B. 11 Franklin St., Rumford  
Martin, Joseph E. 35 Main St., Mexico  
McCormack, Roland L. 12 Bridge St., Norway  
Moore, Beryl M. Oxford  
Nangle, Thomas P. West Paris  
Oestrich, Alfred 89 Congress St., Rumford  
Royal, Albert P., Jr. 82 Maine Ave., Rumford  
Young, John Bethel

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Greene, John A. 96 Congress St., Rumford  
Mills, Nathaniel Harrison  
Pearson, Henry Brownfield  
Stanwood, Harold W. Dixfield  
Stewart, Delbert M. 15 Main St., So. Paris

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Kay, Edwin 31 Frye St., Lewiston

MacDougall, James A.  
Nelson, Chesley W.

303 Penobscot St., Rumford  
121 Main St., Norway

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Perkins, Niles L., Jr. 22 Bramhall St., Portland  
Rowe, Linwood M. 22 Bramhall St., Portland

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Boynton, Willard H. USOM/H&S Div., Box 32, Navy 150,  
c/o FPO, San Francisco, California

#### PENOBSCOT COUNTY

*President* — Clement S. Dwyer, M.D.  
*Secretary* — Frederick C. Emery, M.D.  
*Treasurer* — Benjamin L. Shapero, M.D.

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Adams, Asa C. 68 Main St., Orono  
Adams, Winford C. 255 N. Main St., Brewer  
Babcock, Edward B. 115 Wilson St., Brewer  
Barrett, Robert J., Jr. Cor. Union & James Sts., Bangor  
Blackburn, Nelson P. 489 State St., Bangor  
Blaisdell, Carl E. 47 Broadway, Bangor  
Blaisdell, William B., Jr. 47 Broadway, Bangor  
Blinder, Philip 128 Broadway, Bangor  
Bridges, Donald E. 209 State St., Bangor  
Brod, James J. 51 Grove St., Bangor  
Brown, Eugene E. 57 Summit Ave., Bangor  
Brown, Lloyd 316 State St., Bangor  
Burke, John E. 824 State St., Bangor  
Burke, Paul W. 5 High St., Newport  
Butler, Harry 77 Broadway, Bangor  
Butterfield, Wilfred I. 119 Main St., Lincoln  
Chason, Sidney 128 Broadway, Bangor  
Clement, James D., Jr. 77 Essex St., Bangor  
Clough, Dexter J., 2nd 224 State St., Bangor  
Cornell, Robert C. 118 Forest Ave., Orono  
Coulton, Donald 326 State St., Bangor  
Cross, Harold D. Main Rd. & Summer Sts., Hampden  
Highlands  
Curran, Edward L. 209 State St., Bangor  
Cutler, Lawrence M. 31 Grove St., Bangor  
Desjardins, Richard F. 240 Penobscot Ave., Millinocket  
Dietrich, Mary M. P. O. Box 8, Orrington  
Dulley, Richard V. 187 N. Main St., Brewer  
Dunham, Rand A. P. O. Box 400, E. Millinocket  
Dwyer, Clement S. 205 French St., Bangor  
Emery, Frederick C. 242 Cedar St., Bangor  
Eyerer, Rudolf E. 489 State St., Bangor  
Feeley, J. Robert 316 State St., Bangor  
Fergus, Andrew 128 Broadway, Bangor  
Gaillard, Richard A. 276 State St., Bangor  
Gilman, Herbert C. 240 Penobscot Ave., Millinocket  
Graves, Robert A. Sunset Drive, Orono  
Hall, Walter L. H. 130 Middle St., Old Town  
Hamlin, Irving E. Main St., E. Millinocket  
Hill, Allison K. 113 Somerset St., Bangor  
Houlihan, John S. 209 State St., Bangor  
Hughes, Edward J., Jr. 209 State St., Bangor  
Hutchins, Deane L. Health Dept., Univ. of Maine, Orono  
Irwin, Carl W. 262 State St., Bangor  
Kadi, Francis J. Bangor State Hosp., Bangor  
Kellogg, Robert O. 316 State St., Bangor  
Leddy, Percy A. Main St., Seal Harbor  
Lee, Kong 22 Glenn Dr., Woodbury, L.I., N.Y.  
Lieberman, Arthur N. 180 Broadway, Bangor  
Macdonald, Donald F. 263 State St., Bangor  
Manter, Wilbur B. 1 Fern St., Bangor  
Mason, Peter H. Millinocket Com. Hosp., Millinocket  
McEvoy, Charles D., Jr. 316 State St., Bangor  
McLean, Preston A. 209 State St., Bangor  
McNamara, Wesley C. 8 Lee St., Lincoln  
McQuoid, Robert M. 39 Columbia St., Bangor

Memmelaar, Joseph E.  
Miragliuolo, Leonard G.  
Moulton, Gardner N.  
Munce, Richard T.  
Nesin, Bourcard  
O'Kane, Francis R.  
Osler, Jay K.  
Palmer, Thomas H., Jr.  
Parrot, Hadley  
Pearson, John J.  
Pooler, Harold A.  
Porter, Edward C.  
Purinton, William A.  
Ridlon, Magnus F.  
Ruhlin, Carl W.  
Sewall, Elmer M.  
Shapero, Benjamin L.  
Shubert, Alice J.  
Shubert, William M.  
Shurman, Hans  
Smith, Hugh A.  
Striar, Ronald R.  
Strout, Warren G.  
Sullivan, John R.  
Taylor, H. Lewis  
Thomas, Philip B.  
Todd, Albert C.  
Trowbridge, Mason, Jr.  
Vickers, Martyn A.  
Wadsworth, Richard C.  
Wagner, Samuel L.  
Walker, George R.  
Warren, H. Draper  
Weisz, Hans  
Whitney, Byron V.  
Whitworth, John E.  
Wood, George W., III  
Woodcock, John A.

54 Forest Ave., Bangor  
10 Maple St., Bangor  
5 Grove St., Bangor  
262 State St., Bangor  
10 Water St., Howland  
122 Penobscot Ave., Millinocket  
74 Birch St., Bangor  
316 State St., Bangor  
74 Somerset St., Bangor  
100 S. Main St., Old Town  
Bangor State Hosp., Bangor  
489 State St., Bangor  
15 Ohio St., Bangor  
99 Broadway, Bangor  
205 French St., Bangor  
14 Park St., Orono  
142 Pine St., Bangor  
317 State St., Bangor  
317 State St., Bangor  
10 Spring St., Dexter  
Eastern Maine Gen. Hosp., Bangor  
94 Essex St., Bangor  
205 French St., Bangor  
340 No. Main St., Brewer  
25 Church St., Dexter  
205 French St., Bangor  
185 No. Main St., Brewer  
142 Pine St., Bangor  
268 State St., Bangor  
489 State St., Bangor  
2 Holmes St., Winterport  
128 Broadway, Bangor  
Eastern Maine Gen. Hosp., Bangor  
194 Main St., Lincoln  
280 State St., Bangor  
116 Hammond St. Bangor  
156 No. Main St., Brewer  
35 Second St., Bangor

#### HONORARY MEMBERS

Craig, Allan 28 Baraud Rd., Scarsdale, N. Y.  
Devan, Thomas A. 10245-47th Ave., Corona, L. I., N. Y.  
Hedin, Carl J. Penobscot Terrace, Brewer  
Higgins, George I. 15 Water St., Newport  
Purinton, Watson S. 15 Ohio St., Bangor  
Weatherbee, George B. Main St., Hampden Highlands

#### SENIOR MEMBERS

Ames, Forrest B. 255 Hammond St., Bangor  
Emerson, W. Merritt 131 State St., Bangor  
McNeil, Harry D. 81 Silver Rd., Bangor  
Scribner, Herbert C. 200 Union St., Bangor  
Woodcock, Allan 35 Second St., Bangor

#### AFFILIATE MEMBERS

DeWitt, James C. 1313 Jefferson St., Cuyahoga Falls, Ohio  
Merrill, Urban H. 13 Water St., Newport

#### SERVICE MEMBER

Clough, Herbert T. (Col.) Hq. USAF (AFCSG 12),  
Bldg. T-8, Washington 25, D.C.

#### JUNIOR MEMBER

Babcock, Albert L. 323-7th Ave., Salt Lake City, Utah

#### PISCATAQUIS COUNTY

*President* — George C. Howard, M.D.  
*Secretary-Treasurer* — Isaac Nelson, M.D.

#### ACTIVE MEMBERS

Bradbury, Francis W. 16 E. Main St., Dover-Foxcroft  
Carde, Albert M. 33 Elm St., Milo  
Curtis, John B. 10 High St., Milo

Howard, George C.	Oak St., Guilford
Lightbody, Charles H.	No. Main St., Guilford
Nelson, Isaac	Box 336, Greenville
Nickerson, Norman H.	Greenville
Nielsen, Odd S.	85 Pleasant St., Dexter
Stitham, Linus J.	50 Main St., Dover-Foxcroft
Stuart, Ralph C.	Guilford

#### HONORARY MEMBERS

MacDougal, Wilbur E.	186 Nowell Rd., Bangor
Pritham, Fred J.	Greenville Jct.

#### SENIOR MEMBERS

Bundy, Harvey C.	Milo
Stanhope, Charles N.	South St., Dover-Foxcroft

#### JUNIOR MEMBER

Johnson, James H., Jr.	36 Elm St., Milo
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#### SOMERSET COUNTY

*President* — Edgar J. Smith, M.D.

*Secretary-Treasurer* — Harland G. Turner, M.D.

#### ACTIVE MEMBERS

Amrein, H. Carl	29 Weston Ave., Madison
Ball, Franklin P.	Bingham
Bernard, Albert J.	198 Madison Ave., Skowhegan
Briggs, Paul R.	Hartland
Greenlaw, William A.	129 Main St., Fairfield
Grow, William B.	Central Maine San., Fairfield
Hornstein, Louis S.	220 Water St., Skowhegan
Jordan, W. Edward, Jr.	68 Water St., Skowhegan
Kemezys, Kestutis M.	25 Garfield St., Madison
Laney, Richard P.	50 Water St., Skowhegan
Philbrick, Maurice S.	292 Water St., Skowhegan
Reed, Howard L.	68 Water St., Skowhegan
Smith, Edgar J.	1 Park St., Fairfield
Soroka, Selic	39 High St., Skowhegan
Strickland, Marian L.	Easy St., Canaan
Sullivan, George E.	R.F.D. #1, Fairfield
Szelenyi, Ernest	Central Maine San., Fairfield
Szendey, Andrew M.	26 Gray St., Madison
Turner, Harland G.	Box 38, Norridgewock

#### HONORARY MEMBERS

Humphreys, Ernest D.	91 Main St., Pittsfield
Marston, Henry E.	No. Anson
Webber, Merlon A.	33 Lancey St., Pittsfield

#### SENIOR MEMBER

Lord, Maurice E.	Dees Cabins, Lake Placid, Florida
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#### JUNIOR MEMBER

Lord, Edwin M.	R.F.D. Portland Ave., Old Orchard Beach
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#### WALDO COUNTY

*President* — Raymond L. Torrey, M.D.

*Secretary-Treasurer* — Seth H. Read, M.D.

#### ACTIVE MEMBERS

Albro, Ward A.	27 Northport Ave., Belfast
Caswell, John A.	16 Waldo Ave., Belfast
Cobb, Norman E.	132 Main St., Belfast
Read, Seth H.	15 Church St., Belfast
Stein, Ernest W.	72 Main St., Pittsfield
Temple, George L.	Fahey St., Belfast
Torrey, Raymond L.	Main St., Searsport
Webber, John R.	Dark Harbor

#### HONORARY MEMBERS

Small, Foster C.	169 High St., Belfast
Stevens, Carl H.	18 Franklin St., Belfast

#### WASHINGTON COUNTY

*President* — Rowland B. French, M.D.

*Secretary-Treasurer* — Karl V. Larson, M.D.

#### ACTIVE MEMBERS

Bates, James C.	Eastport
French, Rowland B.	16 Water St., Eastport
Jacob, Donald R.	Princeton
Kazutow, John	P. O. Box 24, Ellsworth
Kiel, Joseph B.	Columbia Falls
Larson, Karl V.	E. Machias
MacBride, Robert G.	25 Washington St., Lubec
McAllister, John W.	39 Water St., Lubec
Mitchell, Hazen C.	Calais
Mundie, Perley J.	32 North St., Calais
Nackley, George N.	1 School St., Machias
Rice, William C.	Main St., Calais
Schlain, Israel	Main St., Jonesport
Sears, Harold G.	Second Ave., Woodland
Webber, Samuel R.	Calais

#### SENIOR MEMBERS

Armstrong, Charles M.	Robbinston
Bennet, DaCosta F.	4 Main St., Lubec
Southworth, John D.	Hartland

#### YORK COUNTY

*President* — Marcel D. Ouellette, M.D.

*Secretary-Treasurer* — Charles W. Kinghorn, M.D.

#### ACTIVE MEMBERS

Anton, Thomas	260 Main St., Biddeford
Bacon, Melvin	122 Main St., Sanford
Belmont, Ralph S.	6 Washington St., Sanford
Berger, Steven	257 Elm St., Biddeford
Binette, Germain A.	331 Main St., Saco
Charest, Leandre R.	314 Alfred St., Biddeford
Cuneo, Kenneth J.	31 Summer St., Kennebunk
Dionne, William E.	75 Main St., Springvale
Downing, J. Robert	35 Summer St., Kennebunk
Drummond, S. Dunton	Bar Mills
Endicott, Ruth E.	16 Main St., Ogunquit
Ficker, Robert F.	Maine St., Kennebunkport
Fortier, Andre P.	68 Foss St., Biddeford
Haas, Carl M.	357 Elm St., Biddeford
Hill, Paul S., Jr.	323 Main St., Saco
Hoffman, Alvin A.	P. O. Box 222, York
Hopkins, Herbert J.	24 Portland Ave., Old Orchard
Houle, Marcel P.	200 Alfred St., Biddeford
Jellerson, Leon R.	34 Winter St., Sanford
Johnston, James S.	York Harbor
LaFond, Robert S.	258 Main St., Saco
Lapirow, Harry	99 Main St., Kennebunk
Leigh, Kenneth E.	Brixham Rd., York
Lesieur, Louis C.	255 Beach St., Saco
Lincourt, Armand S.	122 Main St., Sanford
Lord, George A.	34 Winter St., Sanford
Magauda, Michael M. P.	39 Old Orchard St., Old Orchard Beach
Magocsi, Alexander W.	York
Mahaney, William F.	338 Main St., Saco
Moulton, Marion K.	W. Newfield
Murphy, John J.	84 Portland St., So. Berwick
Myer, John C.	Nasson College, Springvale
O'Sullivan, William B.	331 Main St., Saco
Ouellette, Marcel D.	114 Main St., Sanford
Patane, Joseph M.	256 Alfred St., Biddeford

Perrault, Oscar W.  
 Peterlein, Walter R., Jr.  
 Richards, Carl E.  
 Robert, Roger J. P.  
 Ross, Maurice  
 Roussin, William T.  
 Shaw, G. Patrick  
 Smith, Gerald R.  
 Smith, Oney P.  
 Taylor, Paul E.  
 Turville, Charles S.  
 Vachon, Robert D.  
 Viger, Leopold A.  
 Wolfahrt, Eugene P.

30 South St., Biddeford  
 75 Main St., Springvale  
 34 Winter St., Sanford  
 331 Main St., Saco  
 372 Main St., Saco  
 48 Bacon St., Biddeford  
 357 Elm St., Biddeford  
 Ogunquit  
 Post Rd., Wells  
 9 Wentworth St., Kittery  
 P.O. Box 187, Alfred  
 34 Winter St., Sanford  
 176 Elm St., Biddeford  
 338 Main St., Saco

#### HONORARY MEMBERS

Bunker, Willard H.  
 Davis, Ansel S.  
 Head, Owen B.  
 Larochelle, Joseph R.  
 Sever, James W.  
 Whitney, Ray L.

York Harbor  
 Springvale  
 98 Main St., Sanford  
 42 Bacon St., Biddeford  
 Cape Neddick  
 Cape Porpoise

#### SENIOR MEMBERS

Cobb, Stephen A.  
 Dennett, Carl G.  
 Kinghorn, Charles W.  
 Ross, H. Danforth

34 Winter St., Sanford  
 258 Main St., Saco  
 4 Wentworth St., Kittery  
 34 Winter St., Sanford

# An Alphabetical List of the Members of the Maine Medical Association

The figures in parentheses refer to County Societies as follows: (1) Androscoggin, (2) Aroostook, (3) Cumberland, (4) Franklin, (5) Hancock, (6) Kennebec, (7) Knox, (8) Lincoln-Sagadahoc, (9) Oxford, (10) Penobscot, (11) Piscataquis, (12) Somerset, (13) Waldo, (14) Washington, (15) York.

## A

Adams, Asa C., 68 Main St., Orono (10)  
 Adams, Lester, 9 Knox St., Thomaston (9)  
 Adams, Winford C., 255 North Main St., Brewer (10)  
 Agan, Robert W., 144 State St., Portland (3)  
 Akar, Hamdi, 17 Grove St., Bath (8)  
 Akerberg, Ake, 10 Maple St., South Paris (9)  
 Albert, Armand, 193 Main St., Van Buren (2)  
 Albert, Joseph L., 4 Pleasant St., Fort Kent (2)  
 Albrow, Ward A., 27 Northport Ave., Belfast (13)  
 Allen, Donald E., Sebago Lake (3)  
 Ames, Forrest B., 255 Hammond St., Bangor (10)  
 Amfilo, Basil, 134 Russell St., Lewiston (1)  
 Amrein, H. Carl, 29 Weston Ave., Madison (12)  
 Analis, Harry, 902 Brighton Ave., Portland (3)  
 Anderson, Donald L., 369 Main St., Lewiston (1)  
 Andrews, Anneliese M., Maine Medical Center, Portland (3)  
 Andrews, John F., 20 West St., Boothbay Harbor (8)  
 Ansell, Harvey B., 39 Deering St., Portland (3)  
 Anton, Thomas, 260 Main St., Biddeford (15)  
 Apollonio, Howard L., 22 White St., Rockland (7)  
 Applin, Hilton H., 6 Cumberland St., Brunswick (3)  
 Aranson, Albert, 39 Deering St., Portland (3)  
 Archambault, Philip L., 346 Main St., Lewiston (1)  
 Armstrong, Charles M., Robbinston (14)  
 Asali, Louis A., 29 Deering St., Portland (3)  
 Asherman, Edward G., 131 Chadwick St., Portland (3)  
 Ashley, Alta, Dist. III, Health Office, Augusta (6)  
 Aucoin, Peter B., 87 Congress St., Rumford (9)  
 Aungst, Melvin R., Morneault Building, Fort Kent (2)

## B

Babalian, Leon, 38 Deering St., Portland (3)  
 Babcock, Albert L., 323-7th Ave., Salt Lake City, Utah (10)  
 Babcock, Edward B., 115 Wilson St., Brewer (10)  
 Babcock, Harold S., Castine (5)  
 Bacastow, Merle S., 22 Bramhall St., Portland (3)  
 Bachrach, Louis, 16 Union St., Brunswick (3)  
 Bacon, Melvin, 122 Main St., Sanford (15)  
 Baldini, Elio, 22 Bramhall St., Portland (3)  
 Baldwin, Warren C., 42 Deering St., Portland (3)  
 Ball, Franklin P., Bingham (12)  
 Barker, Nathaniel B. T., 1 South St., Yarmouth (3)  
 Barnard, John M. H., 21 Western Ave., Augusta (6)  
 Barnes, Kirk K., 11 McKee St., Brunswick (3)  
 Barrett, Robert J., Jr., Cor. Union & James Sts., Bangor (10)  
 Barron, Richard E., Main St., Monmouth (6)  
 Barrows, Harris C., 5 Oak St., Boothbay Harbor (8)  
 Bates, James C., Eastport (14)  
 Bauman, Clair S., 159 Silver St., Waterville (6)  
 Bean, H. Richard, 171 Main St., Norway (9)  
 Beaudet, Simon C., 25 Webster St., Lewiston (1)  
 Beckerman, Stanley C., 82 Elm St., Waterville (6)  
 Beecker, Vincent H., 85 Wood St., Lewiston (1)  
 Beegel, Paul M., 80 Goff St., Auburn (1)  
 Beliveau, Bertrand A., 56 Howe St., Lewiston (1)  
 Belknap, Samuel L., Damariscotta (8)  
 Belmont, Ralph S., 6 Washington St., Sanford (15)  
 Bennet, DaCosta F., 4 Main St., Lubec (14)  
 Bennet, Eben T., 49 Deering St., Portland (3)  
 Berger, Steven, 257 Elm St., Biddeford (15)  
 Bernard, Albert J., 198 Madison Ave., Skowhegan (12)  
 Bettle, Ronald A., 32 Federal St., Brunswick (3)  
 Betts, Anthony, 39 Harpswell St., Brunswick (8)  
 Bidwell, Robinson L., 31 Bramhall St., Portland (3)  
 Binette, Germain A., 331 Main St., Saco (15)  
 Bischoffberger, John M., Naples (3)  
 Bisgrove, John G., 165 Park Row, Brunswick (3)  
 Bishop, Lloyd W., 211 Vaughan St., Portland (3)

Black, Paul E., Capt. Naval Air Station, Brunswick (5)  
 Blackburn, Nelson P., 489 State St., Bangor (10)  
 Blaisdell, Carl E., 47 Broadway, Bangor (10)  
 Blaisdell, Elton R., 12 Deering St., Portland (3)  
 Blaisdell, William B., Jr., 47 Broadway, Bangor (10)  
 Blinder, Philip, 128 Broadway, Bangor (10)  
 Blumberg, Edward, Box C, Pownal (3)  
 Bolduc, Jean L., 173 Main St., Waterville (6)  
 Bonney, James H., 229 Vaughan St., Portland (3)  
 Boone, Storer W., 429 Main St., Presque Isle (2)  
 Bostwick, George W., Newcastle (8)  
 Bourassa, Harvey J., 15 Silver St., Waterville (6)  
 Bove, Louis G., 12 Deering St., Portland (3)  
 Bowman, Peter W., P. O. Box C, Pownal (3)  
 Bowne, Hays G., 9A Main St., Farmington (4)  
 Boynton, Willard H., USOM/H&S Div., Box 32, Navy 150, c/o FPO, San Francisco, California (9)  
 Bradbury, Francis W., 16 E. Main St., Dover-Foxcroft (11)  
 Bramhall, Theodore C., 185 Craigie St., Portland (3)  
 Winter address—3531 Mineola Dr., Sarasota, Fla.  
 Branch, Charles F., Central Maine Gen. Hosp., Lewiston (1)  
 Brann, Henry A., 31 Western Ave., Augusta (6)  
 Branson, Sidney R., 37 Main St., South Windham (3)  
 Breard, J. Alfred, 15 Summer St., Waterville (6)  
 Brennan, Thomas V., 99 Hardy St., Presque Isle (2)  
 Bridges, Donald E., 209 State St., Bangor (10)  
 Briggs, Paul R., Hartland (12)  
 Brien, Maurice, 76 Pine St., Lewiston (1)  
 Brinkman, Harry, 47 Perham St., Farmington (4)  
 Brod, James J., 51 Grove St., Bangor (10)  
 Broggi, Frank S., 18 Neal St., Portland (3)  
 Broughton, David S., 1 Pin Oak Lane, Louisville, Ky. (9)  
 Brouwer, Johan, 5 Beech St., Rockland (7)  
 Brown, Douglas H., 548 Shore Rd., Cape Elizabeth (3)  
 Brown, Eugene E., 57 Summit Ave., Bangor (10)  
 Brown, Lloyd, 316 State St., Bangor (10)  
 Brown, Luther A., 13 Deering St., Portland (3)  
 Brown, Stephen S., Mars Hill (2)  
 Brownlow, Bradley E., Blue Hill Mem. Hosp., Blue Hill (5)  
 Buker, Edson B., R. F. D. No. 3, Auburn (1)  
 Bull, Frank B., 72 Church St., Gardiner (6)  
 Bundy, Harvey C., Milo (11)  
 Bunker, Willard H., York Harbor (15)  
 Burke, John E., 824 State St., Bangor (10)  
 Burke, Paul W., 5 High St., Newport (10)  
 Burnett, Claude A. Jr., 59 Deering St., Portland (3)  
 Burns, Robert M., 582 Main St., Westbrook (3)  
 Burr, Charles G., 90 Court St., Houlton (2)  
 Burrage, William C., 57 Deering St., Portland (3)  
 Busch, John J., 105 Elm St., Mechanic Falls (1)  
 Butler, Harry, 77 Broadway, Bangor (10)  
 Butterfield, Wilfred L., 119 Main St., Lincoln (10)

## C

Campbell, Fred G., Box 484, Warren (7)  
 Canal, Ory D., Augusta State Hospital, Augusta (6)  
 Capron, Charles W., 22 Bramhall St., Portland (3)  
 Carde, Albert M., 33 Elm St., Milo (11)  
 Carrier, John W., Central Maine Gen. Hosp., Lewiston (1)  
 Carson, Robert S., 11 McKee St., Brunswick (3)  
 Carton, Arthur K., Market Square, Houlton (2)  
 Casey, William L., 131 State St., Portland (3)  
 Castellanos, Jose, Augusta State Hospital, August (6)  
 Caswell, John A., 16 Waldo Ave., Belfast (13)  
 Chapin, Milan A., 237 Turner St., Auburn (1)  
 Charest, Leandre R., 314 Alfred St., Biddeford (15)  
 Chase, George O., 144 State St., Portland (3)  
 Chase, Philip B., 36 Main St., Farmington (4)  
 Chason, Sidney, 128 Broadway, Bangor (10)  
 Chasse, Richard L., 18 Park St., Waterville (6)

Chatterjee, Manu, 11 McKeen St., Brunswick (3)  
 Chen, Jen-Ti, Cherry Hill Terrace, Waterville (6)  
 Chenery, Frederick L. Jr., Monmouth (1)  
 Christensen, Harry E., South Freeport (3)  
 Ciampi, Louis A., Gray (3)  
 Clapp, Waldo A., 215 College St., Lewiston (1)  
 Clapperton, Gilbert, 300 Main St., Lewiston (1)  
 Clark, Frederick B., 131 State St., Portland (3)  
 Clark, Richard L., P.O. Box 127, Freeport (8)  
 Clarkin, Charles P., 64 Brookside Rd., Portland (3)  
 Clement, James D., Jr., 77 Essex St., Bangor (10)  
 Clough, Dexter J., 2nd, 224 State St., Bangor (10)  
 Clough, Herbert T., (Col.) Hq. USAF (AFCSG 12),  
 Bldg. T-8, Washington 25, D.C. (10)  
 Cloutier, Wilfrid A., 210 Sabattus St., Lewiston (1)  
 Cobb, Norman E., 132 Main St., Belfast (13)  
 Cobb, Stephen A., 34 Winter St., Sanford (15)  
 Coffin, Ernest L., Northeast Harbor (5)  
 Coffin, Silas A., 39 High St., Bar Harbor (5)  
 Cole, Donald P., 45 Deering St., Portland (3)  
 Colley, Maynard B., 14 Main St., Farmington (4)  
 Collins, H. Douglas, Caribou Clinic, Caribou (2)  
 Cook, Aaron, 23 High St., Waterville (6)  
 Cooper, Llewellyn W., 194 Main St., Bar Harbor (5)  
 Cornell, Robert C., 118 Forest Ave., Orono (10)  
 Coulton, Donald, 326 State St., Bangor (10)  
 Covert, Stanley B., Kingfield (4)  
 Cox, William V., 133 Court St., Auburn (1)  
 Cragin, Charles L., 831 Congress St., Portland (3)  
 Craig, Allan, 28 Baraud Rd., Scarsdale, New York (10)  
 Crane, Lawrence, 157 Pine St., Portland (3)  
 Crawford, Albert S., Box 414, Togus (6)  
 Crawford, J. Ramser, 105 Water St., Augusta (6)  
 Crawford, Joseph R., 105 Water St., Augusta (6)  
 Cross, Harold D., Main Rd. & Summer St., Hampden High-  
 lands (10)  
 Crowe, James H., 121 Main St., Ellsworth (5)  
 Cummings, George O., Sr., 47 Deering St., Portland (3)  
 Cummings, George O., Jr., 47 Deering St., Portland (3)  
 Cunco, Kenneth J., 31 Summer St., Kennebunk (15)  
 Curran, Edward L., 209 State St., Bangor (10)  
 Curtis, John B., 10 High St., Milo (11)  
 Cutler, Lawrence M., 31 Grove St., Bangor (10)

## D

Dachslager, Philip, 21 Western Ave., Augusta (6)  
 Dalrymple, Sidney C., So. Great Rd., So. Lincoln, Mass. (8)  
 D'Andrea, Anthony L., 131 State St., Portland (3)  
 Daniels, Donald H., R.R. No. 1, Readfield (3)  
 Darlington, Brinton T., Westwood Rd., Augusta (6)  
 Davidson, David, 235 State St., Portland (3)  
 Davidson, Gisela K., 235 State St., Portland (3)  
 Davies, Lloyd G., 78 Main St., Fryeburg (3)  
 Davis, Ansel S., Springvale (15)  
 Davis, Earle M., 2 School St., Waterville (6)  
 Davis, Harry E., 169 State St., Portland (3)  
 DeCosta, Donald A., Poland Spring (1)  
 Defoe, Garfield G., Dixfield (9)  
 De la Garza, Alexander M., 111 Webster St., Lewiston (1)  
 Denison, John D., 105 Brunswick Ave., Gardiner (6)  
 Dennett, Carl G., 258 Main St., Saco (15)  
 Dennis, Richard H., 33 College Ave., Waterville (6)  
 Dennison, Frederick C., 183 Main St., Thomaston (7)  
 Derry, G. Hermann, 690 Congress St., Portland (3)  
 Desjardins, Arthur U., South Bristol (8)  
 Desjardins, Richard F., 240 Penobscot Ave., Millinocket (10)  
 Devan, Thomas A., 10245-47th Ave., Corona, L. I., N. Y. (10)  
 DeWitt, James C., 1313 Jefferson St., Cuyahoga Falls, Ohio (10)  
 Dienst, Stanley G., 131 Chadwick St., Portland (3)  
 Dietrich, Mary M., P. O. Box 8, Orrington (10)  
 Dionne, Maurice J., 26-28 Cumberland St., Brunswick (3)  
 Dionne, William E., 75 Main St., Springvale (15)  
 Dixon, Walter G., 16 Deering St., Norway (9)  
 Doble, Miriam, 990 Washington St., Bath (8)  
 Doby, Tibor, 131 State St., Portland (3)  
 Dolan, Thomas F., Jr., 50 Union St., Ellsworth (5)  
 Donahue, Clement L., 18 Sweden St., Caribou (2)  
 Donahue, Gerald H., 4 Station St., Presque Isle (2)

Dooley, Francis M., 53 Deering St., Portland (3)  
 Dore, Clarence E., 2 School St., Waterville (6)  
 Dore, Kenneth E., 133 Main St., Fryeburg (3)  
 Dorogi, Louis V., 149 Main St., Freeport (3)  
 Dougherty, John F., 112 Front St., Bath (8)  
 Douphinett, Otis J., 763 Congress St., Portland (3)  
 Downing, J. Robert, 35 Summer St., Kennebunk (15)  
 Drake, Emerson H., 18 Bramhall St., Portland (3)  
 Drexler, James E., Ward Town Rd., Freeport (3)  
 Drummond, S. Dunton, Bar Mills (15)  
 Duffey, Richard V., 187 North Main St., Brewer (10)  
 Duffy, Wallace H., 100 Main St., Farmington (4)  
 Dunham, Carl E., 188 State St., Portland (3)  
 Dunham, Marguerite C., P.O. Box 748, Presque Isle (2)  
 Dunham, Rand A., P.O. Box 400, East Millinocket (10)  
 Dunn, Robert H., Veterans Administration, Togus (6)  
 Dwyer, Clement S., 205 French St., Bangor (10)  
 Dycio, George, 55 Broad St., Auburn (1)  
 Dycio, Mary T., 3 Bayberry Lane, Lewiston (1)  
 Dylberg, Norman E., 323 Main St., Cumberland Mills (3)

## E

Earle, Ralph P., Vinalhaven (7)  
 Earnhardt, Joseph B., 55 Stroudwater St., Westbrook (3)  
 Eastman, Charles W., 15 Millet St., Livermore Falls (4)  
 Eddy, Robert H., 5 Beech St., Rockland (7)  
 Elsmore, Dexter E., 11 Main St., Dixfield (9)  
 Emanuel, Meyer, Veterans Administration, Togus (6)  
 Emerson, W. Merritt, 131 State St., Bangor (10)  
 Emery, Frederick C., 242 Cedar St., Bangor (10)  
 Endicott, Ruth E., 16 Main St., Ogunquit (15)  
 English, Lena M., Veterans Administration, Togus (6)  
 Eppinger, Ernst, 52 Belmont St., Portland (3)  
 Ervin, Edmund N., 2 School St., Waterville (6)  
 Etskovitz, Eli A., Carv Memorial Hospital, Caribou (2)  
 Eyerer, Rudolf E., 489 State St., Bangor (10)

## F

Fagone, Francis A., 312 Congress St., Portland (3)  
 Faucher, Francois J., Grand Isle (2)  
 Feeley, J. Robert, 316 State St., Bangor (10)  
 Fergus, Andrew, 128 Broadway, Bangor (10)  
 Ferguson, Barbara, 80 Goff St., Auburn (1)  
 Ferguson, Franklin F., 22 Bramhall St., Portland (3)  
 Fichtner, Paul A., 781 High St., Bath (4)  
 Ficker, Robert F., Maine St., Kennebunkport (15)  
 Finks, Henry B., 73 Deering St., Portland (3)  
 Fiorica, Gaetano T., 12 Church St., Chisholm (4)  
 Fish, Nicholas, 235 State St., Portland (3)  
 Fisher, Dean H., State House, Augusta (6)  
 Fisher, Samson, 173 Main St., Waterville (6)  
 Fishman, Louis N., 327 Main St., Lewiston (1)  
 Flanders, Merton N., 1 High St., Lewiston (1)  
 Floyd, Paul E., 2 Middle St., Farmington (4)  
 Fogg, C. Eugene, 35 Deering St., Portland (3)  
 Fogg, Philip S., Jr., 173 Pleasant Ave., Portland (3)  
 Fortier, Andre P., 68 Foss St., Biddeford (15)  
 Fortier, Paul J., 111 Webster St., Lewiston (1)  
 Foster, Albert D., 447 Grosvenor Rd., Rochester 10, N.Y. (3)  
 Foster, Thomas A., 131 State St., Portland (3)  
 Fox, Francis H., 83 West St., Portland (3)  
 Freeman, William E., 107 Main St., Yarmouth (3)  
 French, Rowland B., 16 Water St., Eastport (14)  
 Frenette, Francis F., 26 Main St., Washburn (2)  
 Friend, John W., 49 Hampton Ave., Auburn (1)  
 Frost, Harold M., Friendship (7)  
 Frost, Robert A., 93 Summer St., Auburn (1)  
 Fuller, Barbara L., 20 Chestnut St., Rockland (7)

## G

Gaillard, Richard A., 276 State St., Bangor (10)  
 Galen, Robert S., 22 MacMillan Dr., Brunswick (3)  
 Gates, Clifford W., Flaggy Meadow Rd., Gorham (3)  
 Gauvreau, Horace L., 82 Pine St., Lewiston (1)

Gauvreau, Norman O., 78 Pine St., Lewiston (1)  
 Geer, Charles R., 690 Congress St., Portland (3)  
 Geer, George I., Jr., 690 Congress St., Portland (3)  
 Getchell, Ralph A., 690 Congress St., Portland (3)  
 Geyerhahn, George, 73 Deering St., Portland (3)  
 Gibbons, John F., 22 Bramhall St., Portland (3)  
 Giberson, Raymond G., 156 A Academy St., Presque Isle (2)  
 Giddings, Lane, 6 E. Chestnut St., Augusta (6)  
 Giddings, Paul D., 31 Western Ave., Augusta (6)  
 Giesen, Joseph H., 34 Gilman St., Waterville (6)  
 Giguere, Eustache N., 90 Webster St., Lewiston (1)  
 Gilman, Herbert C., 240 Penobscot Ave., Millinocket (10)  
 Gingras, Adolphe J., 99 Water St., Augusta (6)  
 Gingras, Napoleon J., 6 East Chestnut St., Augusta (6)  
 Glassmire, Charles R., 58 Deering St., Portland (3)  
 Goduti, Richard J., 9 Deering St., Portland (3)  
 Goldfarb, Jaime, Box C, Pownal (3)  
 Goldman, Morris E., 524 Main St., Lewiston (1)  
 Good, Philip G., 38 Deering St., Portland (3)  
 Goodof, Irving I., Thayer Hospital, Waterville (6)  
 Goodrich, Blynn O., 165 Main St., Waterville (6)  
 Goodwin, Ralph A., Sr., 56 Denison St., Auburn (1)  
 Goodwin, Ralph A., Jr., 33 Court St., Auburn (1)  
 Gornley, Eugene G., Market Square, Houlton (2)  
 Gould, George I., 79 Main St., Richmond (6)  
 Graves, Robert A., Sunset Drive, Orono (10)  
 Gray, Philip L., Blue Hill (5)  
 Greco, Edward A., 12 Pine St., Portland (3)  
 Green, Ross W., 33 Court St., Auburn (1)  
 Greene, John A., 96 Congress St., Rumford (9)  
 Greene, John P., 19 Sabattus St., Lewiston (1)  
 Greene, Merrill S. F., 466 Main St., Lewiston (1)  
 Greenlaw, William A., 129 Main St., Fairfield (12)  
 Gregory, Frederick J., 16 High St., Caribou (2)  
 Gregory, Philip O., St. Andrews Hosp., Boothbay Harbor (8)  
 Griffin, Carl R., Jr., 69 Townsend Ave., Boothbay Harbor (8)  
 Griffiths, Eugene B., 429 Main St., Presque Isle (2)  
 Grish, Albert J., Box C, Pownal (3)  
 Grow, William B., Central Maine Sanatorium, Fairfield (12)  
 Guillemette, Maurice R., 109 Water St., Augusta (6)  
 Guite, L. Armand, 45 Elm St., Waterville (6)

## H

Haas, Carl M., 357 Elm St., Biddeford (15)  
 Haas, Rudolph, 480 Main St., Lewiston (1)  
 Hall, Walter D., 407 Main St., Rockland (7)  
 Hall, Walter L. H., 130 Middle St., Old Town (10)  
 Hallett, George W., Jr., 72 West St., Portland (3)  
 Hamel, John R., 50 Deering St., Portland (3)  
 Hamilton, Virginia C., 900 Washington St., Bath (8)  
 Hamlin, Irvin E., Main St., East Millinocket (10)  
 Hanley, Daniel F., 58 Federal St., Brunswick (3)  
 Hannigan, Charles A., 85 Goff St., Auburn (1)  
 Hannigan, Margaret H., 85 Goff St., Auburn (1)  
 Hanson, Henry W., Jr., Cumberland Center (3)  
 Harkins, Michael J., 437 Main St., Lewiston (1)  
 Harlow, Edwin W., 177 Main St., Waterville (6)  
 Harper, Harry L., 17 Main St., South Paris (9)  
 Harrison, George J., Market Sq., Houlton (2)  
 Harvey, Thomas G., 59 Mayo St., Caribou (2)  
 Hawkes, Richard S., 47 Deering St., Portland (3)  
 Hawkins, Donald B., Atlantic Ave. and Sea St., Camden (7)  
 Hayward, I. Mead, So. Main St., Caribou (2)  
 Head, Owen B., 98 Main St., Sanford (15)  
 Heath, Parker, Jr., 22 White St., Rockland (7)  
 Hecht, Henry, 326 Stevens Ave., Portland (3)  
 Hedin, Carl J., Penobscot Terrace, Brewer (10)  
 Heifetz, Ralph, 173 State St., Portland (3)  
 Helfrich, Harry M., Jr., 122 Academy St., Presque Isle (2)  
 Helfrich, Nancy R., 48 Third St., Presque Isle (2)  
 Herbert, Walter W., Eastern Mem. Hosp., Ellsworth (5)  
 Herrick, Stanley E., Jr., 12 Deering St., Portland (3)  
 Herring, Leon D., Memorial Dr., Winthrop (6)  
 Herson, Joseph H., 334 E. 25th St., New York, N. Y. (2)  
 Hiebert, Clement A., 18 Bramhall St., Portland (3)  
 Hiebert, Joelle C., Jr., Box 148, Norway (9)  
 Higgins, George F., 122 Academy St., Presque Isle (2)  
 Higgins, George I., 15 Water St., Newport (10)

Hill, Allison K., 113 Somerset St., Bangor (10)  
 Hill, Douglas R., 855 Sawyer St., South Portland (3)  
 Hill, Frederick T., Thayer Hospital, Waterville (6)  
 Hill, Howard F., 33 College Ave., Waterville (6)  
 Hill, Kevin, 33 College Ave., Waterville (6)  
 Hill, Paul S., Jr., 323 Main St., Saco (15)  
 Hinckley, Harris, 331 Cottage Rd., South Portland (3)  
 Hirschberger, Celia, 44 Main St., Waterville (6)  
 Hirshler, Max, 25 Bardwell St., Lewiston (1)  
 Hochschild, Hugo, 33 Main St., Thomaston (7)  
 Hoffman, Alvin A., P. O. Box 222, York (15)  
 Hogan, Chester F., 62 Main St., Houlton (2)  
 Holt, C. Lawrence, 27 Deering St., Portland (3)  
 Hopkins, Herbert J., 24 Portland Ave., Old Orchard (15)  
 Hopping, John S., R.F.D. No. 2, Union (7)  
 Hornberger, H. Richard, 2 School St., Waterville (6)  
 Hornstein, Louis S., 220 Water St., Skowhegan (12)  
 Horsman, Donald H., 50 Goff St., Auburn (1)  
 Houle, Marcel P., 200 Alfred St., Biddeford (15)  
 Houlihan, John S., 209 State St., Bangor (10)  
 Howard, George C., Oak St., Guilford (11)  
 Howard, Henry M., 105 Franklin St., Rumford (9)  
 Hsu, Theodore S., 14 High St., Ellsworth (5)  
 Hubbard, Roswell E., Waterford (9)  
 Hudson, Henry A., R.F.D. #1, West Bridgton (3)  
 Hughes, Edward J. Jr., 20<sup>th</sup> State St., Bangor (10)  
 Humphreys, Ernest D., 91 Main St., Pittsfield (12)  
 Hunter, Albert L., Knox County Gen. Hosp., Rockland (7)  
 Huntress, Roderick L., 988 Sawyer St., South Portland (3)  
 Hurd, Allan C., 72 Church St., Gardiner (6)  
 Hutchins, Deane L., Health Department, Univ. of Maine, Orono (10)

## I

Irwin, Carl W., 262 State St., Bangor (10)  
 Ives, Howard R., 131 Chadwick St., Portland (3)

## J

Jackler, Jacob M., 14 Gilman St., Waterville (6)  
 Jackson, Norman M., 89 Congress St., Rumford (9)  
 Jacob, Donald R., Princeton (14)  
 Jacobson, Payson B., 295 Brighton Ave., Portland (3)  
 James, Chakmakis, 47 Howe St., Lewiston (1)  
 James, John A., 117 Goff St., Auburn (1)  
 Jameson, C. Harold, Medical Arts Building, Rockland (7)  
 Jellerson, Leon R., 34 Winter St., Sanford (15)  
 Jennings, Richard K., Univ. of Mass., Amherst, Mass. (5)  
 Johnson, Albert C., 131 Chadwick St., Portland (3)  
 Johnson, Gordon N., P. O. Box 86, Houlton (2)  
 Johnson, Henry P., 32 Deering St., Portland (3)  
 Johnson, James H., Jr., 36 Elm St., Milo (11)  
 Johnson, Oscar R., 18 Deering St., Portland (3)  
 Johnson, R. Paul, Main St., Fort Kent (2)  
 Johnston, James S., York Harbor (15)  
 Jones, Paul A., Sr., Union (7)  
 Jones, Paul A., Jr., 2 School St., Waterville (6)  
 Joost, Arthur M., Jr., P. O. Box B, Bucksport (5)  
 Jordan, W. Edward, Jr., 68 Water St., Skowhegan (12)

## K

Kadi, Francis J., Bangor State Hospital, Bangor (10)  
 Kagan, Samuel H., 283 Water St., Augusta (6)  
 Kay, Edwin, 31 Frye St., Lewiston (9)  
 Kazutow, John, P. O. Box 24, Ellsworth (14)  
 Kellogg, Robert O., 316 State St., Bangor (10)  
 Kemezis, Kestutis M., 25 Garfield St., Madison (12)  
 Kent, Stanley W., 42 Deering St., Portland (3)  
 Kershner, Warren E., 57 Green St., Bath (8)  
 Kibbe, Frank W., R.F.D., Lincolnville (7)  
 Kiel, Joseph B., Columbia Falls (14)  
 Kimball, Herrick C., P. O. Box 372, Fort Fairfield (2)  
 Kinder, Edward L., Jr., 1027 Washington St., Bath (8)  
 King, Merrill J., Sr., 22 White St., Rockland (7)  
 King, Merrill J., Jr., 22 White St., Rockland (7)

Kinghorn, Charles W., 4 Wentworth St., Kittery (15)  
 Kirk, William V., Eagle Lake (2)  
 Knickerbocker, Charles H., 15 High St., Bar Harbor (5)  
 Knowles, Robert M., 49 Deering St., Portland (3)  
 Konecki, John T., St. Mary's Hospital, Lewiston (1)  
 Kopfmann, Harry, Deer Isle (5)  
 Kramer, Henry F., Caribou Clinic, Caribou (2)  
 Kretzing, Harold G., 331 Veranda St., Portland (3)  
 Kudisch, Leonidas B., 11 Franklin St., Rumford (9)

## L

Labbe, Onil B., Van Buren (2)  
 LaFlamme, Paul J., 106 Russell St., Lewiston (1)  
 LaFond, Robert S., 258 Main St., Saco (15)  
 Landwehr, George R., 111 Water St., Augusta (6)  
 Lane, Russell M., Water St., Blue Hill (5)  
 Laney, Richard P., 50 Water St., Skowhegan (12)  
 Langer, Ella, State House, Augusta (6)  
 Lansing, Peter F., 16 Macomber Ave., Augusta (6)  
 Lape, C. Philip, 131 Chadwick St., Portland (3)  
 Lapirow, Harry, 99 Main St., Kennebunk (15)  
 Lappin, John J., 171 State St., Portland (3)  
 Larochelle, Joseph R., 42 Bacon St., Biddeford (15)  
 Larrabee, Charles F., 48 Mt. Desert St., Bar Harbor (5)  
 Larson, Karl V., East Machias (14)  
 Laughlin, K. Alexander, 201 State St., Portland (3)  
 Lawry, Oram R., Jr., 96 Limerock St., Rockland (7)  
 Leary, Gerald C., 144 State St., Portland (3)  
 Leddy, Percy A., Main St., Seal Harbor (10)  
 Lee, Kong, 22 Glenn Dr., Woodbury, L.I., N.Y. (10)  
 Leigh, Kenneth E., Brixham Rd., York (15)  
 Leighton, Wilbur F., 192 State St., Portland (3)  
 Leiter, Laban W., 175 Vaughan St., Portland (3)  
 Leitman, Reuben, 188 Sabattus St., Lewiston (1)  
 Lenfest, Stanley R., Waldoboro (8)  
 Lepore, Anthony E., 72 Church St., Gardiner (6)  
 Lesieur, Louis C., 255 Beach St., Saco (15)  
 Levesque, Romeo J., Frenchville (2)  
 Libby, Harold E., 310 Main St., Westbrook (3)  
 Lichter, Horacio A., 54 Pine St., Lewiston (1)  
 Lidstone, Frederick B., 117 Goff St., Auburn (1)  
 Lieberman, Arthur N., 180 Broadway, Bangor (10)  
 Lightbody, Charles H., No. Main St., Guilford (11)  
 Lincoln, John R., 22 Bramhall St., Portland (3)  
 Lincourt, Armand S., 122 Main St., Sanford (15)  
 Loewenstein, George, Chebeague Island (7)  
 Winter Address — Aripeka, Florida  
 Logan, G. E. C., 131 State St., Portland (3)  
 Lombard, Reginald T., 793 Main St., South Portland (3)  
 Lord, Edwin M., Portland Ave., Old Orchard Beach (12)  
 Lord, George A., 34 Winter St., Sanford (15)  
 Lord, Maurice E., Dees Cabins, Lake Placid, Florida (12)  
 Lorimer, Robert V., 148 State St., Portland (3)  
 Love, Robert B., 97 Main St., Gorham (3)  
 Lovely, David K., 46 Deering St., Portland (3)  
 Lynn, Geraldine, 188 Russell St., Lewiston (1)

## M

MacBride, Robert G., 25 Washington St., Lubec (14)  
 Macdonald, Donald F., 263 State St., Bangor (10)  
 MacDougal, Wilbur E., 186 Nowell Rd., Bangor (11)  
 MacDougall, James A., 303 Penobscot St., Rumford (9)  
 Mack, Francis X., 144 State St., Portland (3)  
 MacVane, William L., Jr., 211 State St., Portland (3)  
 Madigan, John B., Houlton (2)  
 Magauddy, Michael M. P., 39 Old Orchard St., Old Orchard Beach (15)  
 Magocsi, Alexander W., York (15)  
 Mahaney, William F., 338 Main St., Saco (15)  
 Maier, Paul, 723 Congress St., Portland (3)  
 Maltby, George L., 31 Bramhall St., Portland (3)  
 Manol, Jack, 157 Pine St., Portland (3)  
 Manter, Wilbur B., 1 Fern St., Bangor (10)  
 Marquardt, Matthias, Augusta State Hospital, Augusta (6)  
 Marshall, Donald F., 142 High St., Portland (3)  
 Marshall, Joseph A., 177 Main St., Waterville (6)

Marshall, Richard A., 22 Bramhall St., Portland (3)  
 Marsters, David W., Phillips (4)  
 Marston, Henry E., North Anson (12)  
 Marston, Paul C., Kezar Falls (3)  
 Martel, Cyprien L., Jr., 91 Bartlett St., Lewiston (1)  
 Martin, Joseph E., 35 Main St., Mexico (9)  
 Martin, Ralf, 131 Chadwick St., Portland (3)  
 Martin, Thomas A., 157 Pine St., Portland (3)  
 Mason, Peter H., Millinocket Com. Hosp., Millinocket (10)  
 Mathews, Hugh J., Jr., 345 Water St., Gardiner (6)  
 Matthews, Edward C., 131 Chadwick St., Portland (3)  
 Mautner, Hans V., 44 Lafayette St., Yarmouth (3)  
 Mazzone, Giovanni, 487 Stevens Ave., Portland (3)  
 Melendy, Oakley A., Doctors Park, 89 Hospital St., Augusta (6)  
 Melkis, Andrew, Box C, Pownal (3)  
 Melnick, Jacob, 333 Congress St., Portland (3)  
 Memmelaar, Joseph E., 54 Forest Ave., Bangor (10)  
 Mendes, Joseph M., 5 School St., Lisbon Falls (1)  
 Mendros, John G., 111 Webster St., Lewiston (1)  
 Merrill, Urban H., 13 Water St., Newport (10)  
 Methot, Frank P., 54 Pine St., Lewiston (1)  
 Michaud, Joseph C., 6377 Eldredge Rd., Bedford Heights, Ohio (6)  
 Milazzo, John, 42 Elm St., Auburn (1)  
 Millard, Kathleen M. A., Windham Ctr. Rd., Windham (3)  
 Miller, Clark F., 46 Madison St., Auburn (1)  
 Miller, Hudson R., 11 Turner St., Auburn (1)  
 Miller, Thor, 752 Main St., Westbrook (3)  
 Milliken, Howard H., 105 Second St., Hallowell (6)  
 Millington, Paul A., 44 Mountain St., Camden (7)  
 Mills, Nathaniel, Harrison (9)  
 Miragliuolo, Leonard G., 10 Maple St., Bangor (10)  
 Mitchell, Hazen C., Calais (14)  
 Möhlar, Robert G., 11 McKean St., Brunswick (3)  
 Monaghan, Stephen E., 157 Pine St., Portland (3)  
 Monkhouse, William A., 131 State St., Portland (3)  
 Monsivais, Alfredo, Augusta State Hospital, Augusta (6)  
 Moore, Beryl M., Oxford (9)  
 Moore, Valentine J., Thayer Hospital, Waterville (6)  
 Morin, Gerard L., 104 Ash St., Lewiston (1)  
 Morin, Harry F., 905 Middle St., Bath (8)  
 Morissette, Russell A., 460 Main St., Lewiston (1)  
 Morris, Craig W., 50 Bangor St., Augusta (6)  
 Morrison, Alvin A., 57 Deering St., Portland (3)  
 Morrison, James B., Main St., Ashland (2)  
 Morse, Edward K., 22 White St., Rockland (7)  
 Moulton, Albert W., 180 State St., Portland (3)  
 Moulton, Albert W., Jr., Commanding Officer, 173rd Medical Battalion, Fort Dix, N.J. (3)  
 Moulton, Gardner N., 5 Grove St., Bangor (10)  
 Moulton, Marion K., West Newfield (15)  
 Munce, Richard T., 262 State St., Bangor (10)  
 Mundie, Perley J., 32 North St., Calais (14)  
 Murphy, John J., 84 Portland St., South Berwick (15)  
 Myer, John C., Nason College, Springvale (15)

## Mc

McAdams, William R., 723 Congress St., Portland (3)  
 McAllister, John W., 39 Water St., Lubec (14)  
 McCann, Eugene C., 49 Deering St., Portland (3)  
 McCormack, Roland L., 12 Bridge St., Norway (9)  
 McCormac, Philip H., 188 State St., Portland (3)  
 McEvoy, Charles D., Jr., 316 State St., Bangor (10)  
 McFarland, Edward A., 159 Maine St., Brunswick (3)  
 McIntire, Barron F., Jr., 13 W. Elm St., Yarmouth (3)  
 McIntyre, John D., 50 Union St., Ellsworth (5)  
 McKay, Roland L., P.O. Box 265, Augusta (6)  
 McLaughlin, Clarence R., 345 Water St., Gardiner (6)  
 McLaughlin, Ivan E., 345 Water St., Gardiner (6)  
 McLean, E. Allan, 29 Deering St., Portland (3)  
 McLean, Preston A., 209 State St., Bangor (10)  
 McLellan, William A., 87 Chestnut St., Camden (7)  
 McManamy, Eugene P., 72 West St., Portland (3)  
 McMichael, Morton, 73 Deering St., Portland (3)  
 McNamara, Wesley C., 8 Lee St., Lincoln (10)  
 McNeil, Harry D., 81 Silver Rd., Bangor (10)  
 McQuillan, Arthur H., 177 Main St., Waterville (6)  
 McQuoid, Robert M., 39 Columbia St., Bangor (10)

## N

Nackley, George N., 1 School St., Machias (14)  
 Nadeau, J. Paul, 91 Pine St., Lewiston (1)  
 Nadeau, Lawrence A., 41 Sherbrooke Ave., Lewiston (1)  
 Namtze, Chan, Major, MC Ireland Army Hosp., Fort Knox, Kentucky (1)  
 Nangle, Thomas P., West Paris (9)  
 Nelson, Chesley W., 121 Main St., Norway (9)  
 Nelson, Isaac, Box 336, Greenville (11)  
 Nesin, Bourcard, 10 Water St., Howland (10)  
 Newcomb, Charles H., Clinton (6)  
 Nichols, Arthur A., Edgecomb (8)  
 Nickerson, Norman H., Greenville (11)  
 Nielsen, Odd S., 85 Pleasant St., Dexter (11)

## O

Oceretko, Arkadij, 37 Court St., Bath (8)  
 O'Connell, George B., 11 Lisbon St., Lewiston (1)  
 O'Connor, Francis J., 4 Woodlawn St., Augusta (6)  
 O'Donnell, Eugene E., 32 Deering St., Portland (3)  
 Oestrich, Alfred, 89 Congress St., Rumford (9)  
 Ohler, Robert L., Veterans Administration, Togus (6)  
 O'Kane, Francis R., 122 Penobscot Ave., Millinocket (10)  
 Olmsted, Burton L., 73 Deering St., Portland (3)  
 O'Meara, Edward S., Eastern Mem. Hosp., Ellsworth (5)  
 Onat, Mustafa V., St. George (7)  
 Orbeton, Everett A., 131 Chadwick St., Portland (3)  
 Osborne, John R., Veterans Adm., Togus (2)  
 Osher, Harold L., 131 Chadwick St., Portland (3)  
 Osler, Jay K., 74 Birch St., Bangor (10)  
 O'Sullivan, William B., 331 Main St., Saco (15)  
 Ottum, Alvin E., 148 State St., Portland (3)  
 Ouellette, Benoit, 77 Main St., Fort Kent (2)  
 Ouellette, Marcel D., 114 Main St., Sanford (15)

## P

Page, Rosario A., 20 Sweden St., Caribou (2)  
 Palmer, Thomas H., Jr., 316 State St., Bangor (10)  
 Papadopoulos, George, Provincial Mental Hospital, Essondale, B.C., Canada (6)  
 Parcher, George, 75 Main St., Ellsworth (5)  
 Parrot, Hadley, 74 Somerset St., Bangor (10)  
 Patane, Joseph M., 256 Alfred St., Biddeford (15)  
 Patterson, James, 1 Bay Rd., South Portland (3)  
 Patton, Charles H., Jr., 11 McKeen St., Brunswick (3)  
 Pawle, Robert H., 8 Walcott Ave., Falmouth (3)  
 Pearson, Henry, Brownfield (9)  
 Pearson, John J., 100 So. Main St., Old Town (10)  
 Peddie, Harry M. K., 23 Western Ave., Augusta (6)  
 Pendleton, Arthur D., 3 Green St., Fort Fairfield (2)  
 Pennoyer, Douglass C., 112 Vaughan St., Portland (3)  
 Penta, Walter E., 316 Woodford St., Portland (3)  
 Perkins, Niles L., Jr., 22 Bramhall St., Portland (9)  
 Perrault, Oscar W., 30 South St., Biddeford (15)  
 Perri, John A., 331 Veranda St., Portland (3)  
 Peterlein, Walter R., Jr., 75 Main St., Springvale (15)  
 Petterson, Herman C., Chebeague Island (3)  
 Pfeiffer, Paul H., 14 Gilman St., Waterville (6)  
 Philbrick, Maurice S., 292 Water St., Skowhegan (12)  
 Philpot, Van B., Jr., Cary Memorial Hospital, Caribou (2)  
 Pines, Philip, Maine St., Limestone (2)  
 Platt, Anna, Beauchamp Rd., Rockport (7)  
 Winter Address — 110 Manatee Rd., Belleair, Clearwater, Florida  
 Plimpton, Jay R., 283 Water St., Augusta (6)  
 Pogue, Jackson S., 529 Gilmore Ave., Trafford, Pa. (3)  
 Polimer, Irving J., 235 State St., Portland (3)  
 Polisner, Saul R., 143 Vaughan St., Portland (3)  
 Pomerleau, Ovid F., 179 Main St., Waterville (6)  
 Pomerleau, Rodolphe J. F., 27 Main St., Waterville (6)  
 Pooler, Harold A., State Hospital, Bangor (10)  
 Porter, Edward C., 489 State St., Bangor (10)  
 Porter, Joseph E., 22 Bramhall St., Portland (3)  
 Potts, Ronald S., Central Maine Gen. Hosp., Lewiston (1)  
 Poulin, Albert A., Cherry Hill Dr., Waterville (6)

Poulin, James E., 177 Main St., Waterville (6)  
 Powell, Ralph C., Damariscotta (8)  
 Pratt, George L., 7 Main St., Farmington (4)  
 Pratt, Harold S., Livermore Falls (1)  
 Pratt, Loring W., 177 Main St., Waterville (6)  
 Price, Richard D., E. Presque Isle Rd., Caribou (2)  
 Priest, Maurice A., 108 S. Stone St., Deland, Fla. (6)  
 Pritham, Fred J., Greenville Junction (11)  
 Proctor, Ray A., Garden Circle, Caribou (2)  
 Proctor, Thomas E., Boothbay Harbor (8)  
 Proudian, Paul O., 776 Main St., Westbrook (3)  
 Proulx, Harvey J., 92 Pine St., Lewiston (1)  
 Provost, Helen C., 48 Green St., Augusta (6)  
 Provost, Pierre E., 48 Green St., Augusta (6)  
 Purinton, Watson S., 15 Ohio St., Bangor (10)  
 Purinton, William A., 15 Ohio St., Bangor (10)

## R

Rand, Carleton H., 219 Oak St., Lewiston (1)  
 Rando, Joseph J., 111 Webster St., Lewiston (1)  
 Ray, Ferris S., 131 Chadwick St., Portland (3)  
 Read, Seth H., 15 Church St., Belfast (13)  
 Reed, Howard L., 68 Water St., Skowhegan (12)  
 Reed, James W., 18 Main St., Farmington (4)  
 Reel, John J., 59 So. Front St., Richmond (6)  
 Reeves, Edward L., 179 Sabattus St., Lewiston (1)  
 Reeves, Helene M., 179 Sabattus St., Lewiston (1)  
 Reynolds, Arthur P., 29 Second St., Presque Isle (2)  
 Reynolds, John F., 216 Main St., Waterville (6)  
 Reynolds, Ralph L., 216 Main St., Waterville (6)  
 Rice, William C., Main St., Calais (14)  
 Richards, A. Dewey, 11 Gage St., Bridgton (3)  
 Richards, Carl E., 34 Winter St., Sanford (15)  
 Richards, Lee W., Jr., 21 Western Ave., Augusta (6)  
 Rideout, Samuel, 3 Green St., Fort Fairfield (2)  
 Ridlon, Magnus F., 99 Broadway, Bangor (10)  
 Risley, Edward H., P. O. Box 143, Prides Crossing, Mass. (6)  
 Robert, Roger J. P., 331 Main St., Saco (15)  
 Robertson, George J., 33 College Ave., Waterville (6)  
 Robinson, Carl M., Waites Landing, R. 99, Portland (3)  
 Robinson, Hugh P., 131 Chadwick St., Portland (3)  
 Rock, Daniel A., 477 Main St., Lewiston (1)  
 Root, John A., 22 White St., Rockland (7)  
 Ross, H. Danforth, 34 Winter St., Sanford (15)  
 Ross, Maurice, 372 Main St., Saco (15)  
 Roussin, William T., 48 Bacon St., Biddeford (15)  
 Rowe, Daniel M., Kirkwood Rd., Scarborough Beach (3)  
 Rowe, Gunther H., 42 Main St., Livermore Falls (4)  
 Rowe, Linwood M., 22 Bramhall St., Portland (9)  
 Royal, Albert P., Jr., 82 Maine Ave., Rumford (9)  
 Ruhlin, Carl W., 205 French St., Bangor (10)  
 Runyon, William N., 283 Water St., Augusta (6)  
 Russell, Daniel F. D., Leeds (1)  
 Russell, Robert F., Penobscot (5)

## S

Sager, George F., 18 Bramhall St., Portland (3)  
 Sanders, Stephen W., 120 Main St., Winthrop (6)  
 Santoro, Domenico A., 43 Deering St., Portland (3)  
 Sapiro, Howard M., 171 State St., Portland (3)  
 Saunders, Allen I., Ferry Rd., R.F.D. 2, Augusta (6)  
 Savage, Richard L., 4 Em St., Fort Kent (2)  
 Sawyer, Howard P., Jr., 22 Bramhall St., Portland (3)  
 Schlain, Israel, Main St., Jonesport (14)  
 Schmidt, Lorrimer M., Veterans Administration, Togus (6)  
 Schumacher, William E., 14 Westwood Rd., MD "B", Augusta (6)  
 Schwartz, Carol, 3 Deering St., Portland (3)  
 Schwarz, Harald J., Sisters Hospital, Waterville (6)  
 Scolten, Adrian H., 32 Deering St., Portland (3)  
 Scribner, Herbert C., 200 Union St., Bangor (10)  
 Sears, Harold G., Second Ave., Woodland (14)  
 Seligman, Morris J., Veterans Administration, Togus (6)  
 Selvage, Irving L., Jr., 22 Bramhall St., Portland (3)  
 Senenky, Joseph P., Augusta State Hospital, August (6)  
 Sever, James W., Cape Neddick (15)

Sewall, Elmer M., 14 Park St., Orono (10)  
 Sewall, Kenneth W., 2 School St., Waterville (6)  
 Shannon, Charles E. G., 9 Park St., Waterville (6)  
 Shapero, Benjamin L., 142 Pine St., Bangor (10)  
 Shapiro, Morrill, 29 Deering St., Portland (3)  
 Shaw, G. Patrick, 357 Elm St., Biddeford (15)  
 Shaw, John H., 8 Wheeler Park, Brunswick (3)  
 Shelton M. Tieche, 61 Winthrop St., Augusta (6)  
 Shems, Albert, 487 Main St., Lewiston (1)  
 Shields, Daniel R., 369 Main St., Lewiston (1)  
 Shippee, James N., 122 Main St., Winthrop (6)  
 Shubert, Alice J., 317 State St., Bangor (10)  
 Shubert, William M., 317 State St., Bangor (10)  
 Shurman, Hans, 10 Spring St., Dexter (10)  
 Sidwell-Thompson, Doris M., R.F.D. Whittier Rd., W. Ossipee, N. H. (3)  
 Simpson, Margaret R., Box 275, Togus (6)  
 Skillin, Charles E., 690 Congress St., Portland (3)  
 Skillin, Frederick W., 69 So. High St., Bridgton (3)  
 Sleeper, Francis H., Augusta State Hospital, Augusta (10)  
 Small, Foster C., 169 High St., Belfast (13)  
 Smith, Carroll H., Box 967, Presque Isle (2)  
 Smith, Edgar J., 1 Park St., Fairfield (12)  
 Smith, Gerald R., Ogunquit (15)  
 Smith, Hugh A., Eastern Maine Gen. Hosp., Bangor (10)  
 Smith, Jacob, 118 Front St., Bath (8)  
 Smith, Joseph I., 118 Front St., Bath (8)  
 Smith, Kenneth E., Veterans Administration, Togus (6)  
 Smith, Margaret S., Box 967, Presque Isle (2)  
 Smith, Oney P., Post Rd., Wells (15)  
 Somerville, Robert B., 45 Hillside St., Presque Isle (2)  
 Somerville, Wallace B., Mars Hill (2)  
 Sommerfeld, Kurt A., 5 Brunswick Ave., Gardiner (6)  
 Soroka, Selic, 39 High St., Skowhegan (12)  
 Soule, Gilmore W., 22 White St., Rockland (7)  
 Southern, Edward M., 34 Gilman St., Waterville (6)  
 Southworth, John D., Hartland (14)  
 Sowles, Horace K., 413 Blackstrap Rd., Falmouth (3)  
 Spear, William, 107 Main St., Lisbon Falls (1)  
 Spellman, Francis A., Veterans Administration, Togus (6)  
 Stanhope, Charles N., South St., Dover-Foxcroft (11)  
 Stanwood, Harold W., Dixfield (9)  
 Stebbins, Arthur P., 131 State St., Portland (3)  
 Steele, Charles W., 472 Main St., Lewiston (1)  
 Stein, Ernest W., 72 Main St., Pittsfield (13)  
 Stephenson, Richard B., 131 Chadwick St., Portland (3)  
 Stetson, Rufus E., Damariscotta (8)  
 Stevens, Carl H., 18 Franklin St., Belfast (13)  
 Stevens, Theodore M., 148 State St., Portland (3)  
 Stewart, Delbert M., 15 Main St., South Paris (9)  
 Stinchfield, Allan J., P.O. Box 343, Augusta (6)  
 Stitham, Linus J., 50 Main St., Dover-Foxcroft (11)  
 Stocks, Joseph F., 67 Silver St., Waterville (6)  
 Storer, Daniel P., 12 Deering St., Portland (3)  
 Striar, Ronald R., 94 Essex St., Bangor (10)  
 Strickland, Marian L., Easy St., Canaan (12)  
 Strout, Warren G., 205 French St., Bangor (10)  
 Stuart, Ralph C., Guilford (11)  
 Sturtevant, Vaughn R., 33 College Ave., Waterville (6)  
 Sullivan, George E., R. F. D. 1, Fairfield (12)  
 Sullivan, John R., 340 North Main St., Brewer (10)  
 Suyama, Eji, 58 W. Main St., Ellsworth (5)  
 Sweatt, Linwood A., 48 Drummond St., Auburn (1)  
 Swett, Alfred E., 308 Minot Ave., Auburn (1)  
 Swett, Clyde I., 18 Sherman St., Island Falls (2)  
 Sylvester, Stanley B., 1377 Washington Ave., Portland (3)  
 Selenyi, Ernest, Central Maine Sanatorium, Fairfield (12)  
 Szendey, Andrew M., 26 Gray St., Madison (12)

## T

Tabachnick, Henry M., 110 Park Ave., Portland (3)  
 Tashiro, Sabro, 181 Highland Ave., Gardiner (6)  
 Taylor, H. Lewis, 25 Church St., Dexter (10)  
 Taylor, Paul E., 9 Wentworth St., Kittery (15)  
 Taylor, William E., Providence Ave., Falmouth Foreside (3)  
 Tchao, Jou S., 82 Pine St., Lewiston (1)  
 Telfeian, Alphonse, 690 Congress St., Portland (3)  
 Temple, George L., Fahey St., Belfast (13)

Tetreau, William J., 144 Spring St., Portland (3)  
 Thacher, Henry C., 117 Goff St., Auburn (1)  
 Thaxter, Langdon T., Route 100, Portland (3)  
 Thegen, W. Edward, Elm St., Bucksport (5)  
 Thomas, Philip B., 205 French St., Bangor (10)  
 Thompson, Philip P., Jr., 131 Chadwick St., Portland (3)  
 Tibbetts, Otis B., 181 Gamage Ave., Auburn (1)  
 Titherington, John B., 209 State St., Portland (3)  
 Todd, Albert C., 185 North Main St., Brewer (10)  
 Torrey, Marcus A., 75 State St., Ellsworth (5)  
 Torrey, Raymond L., Main St., Searsport (13)  
 Tougas, Raymond A., 8 Cumberland St., Brunswick (3)  
 Tounge, Harry G., Jr., 12 Union St., Camden (7)  
 Tournant, Camille, 111 Pine St., Lewiston (1)  
 Toussaint, Leonid G., P. O. Box 9, Fort Kent (2)  
 Towne, Charles E., 18 Common St., Waterville (6)  
 Tracy, Mary J., Bristol Rd., Damariscotta (8)  
 Trowbridge, Mason, Jr., 142 Pine St., Bangor (10)  
 Turcotte, Guy N., 38 Deering St., Portland (3)  
 Turgeon, Raphael F., 836 Main St., Westbrook (3)  
 Turnbull, Elliott D., 301 Allen Ave., Portland (3)  
 Turner, Harland G., Box 38, Norridgewock (12)  
 Turville, Charles S., P.O. Box 187, Alfred (15)

## U

Urjanis, Janis, Box C, Pownal (3)

## V

Vachon, Robert D., 34 Winter St., Sanford (15)  
 Van Lonkhuyzen, Maurice, 31 Bramhall St., Portland (3)  
 Veilleux, Lucien F., 173 Main St., Waterville (6)  
 Ventimiglia, William A., 117 Hunt Dr., Fayetteville, N. Y. (3)  
 Vickers, Martyn A., 268 State St., Bangor (10)  
 Viger, Leopold A., 176 Elm St., Biddeford (15)  
 Vogell, Frederick C., So. Main St., Caribou (2)

## W

Wadsworth, Richard C., 489 State St., Bangor (10)  
 Waggoner, Gerard M., RMS, Fort Williams, So. Portland (3)  
 Wagner, Samuel L., 2 Holmes St., Winterport (10)  
 Wakefield, Robert D., St. Mary's Hospital, Lewiston (1)  
 Walker, George R., 128 Broadway, Bangor (10)  
 Ward, John V., 131 State St., Portland (3)  
 Ward, William W., 76 Limerock St., Rockland (7)  
 Warren, H. Draper, Eastern Maine Gen. Hosp., Bangor (10)  
 Wasgatt, Wesley N., 41 Talbot Ave., Rockland (7)  
 Waterman, Dorothy, Waldoboro (7)  
 Waterman, Richard, Waldoboro (7)  
 Weatherbee, George B., Main St., Hampden Highlands (10)  
 Weaver, Michael L., 36 Federal St., Brunswick (3)  
 Webber, Isaac M., 29 Deering St., Portland (3)  
 Webber, John R., Dark Harbor (13)  
 Webber, Merlon A., 33 Lancey St., Pittsfield (12)  
 Webber, Samuel R., Calais (14)  
 Webber, Wallace E., 297 Main St., Lewiston (1)  
 Webber, Wedgwood P., 376 Main St., Lewiston (1)  
 Weeks, DeForest, 1 Lantern Lane, Cumberland Foreside (3)  
 Weisz, Hans, 194 Main St., Lincoln (10)  
 Wellington, J. Foster, 396 Brighton Ave., Portland (3)  
 Weltman, Joseph S., Veterans Administration, Togus (6)  
 Westmeyer, Marion W., 32 Federal St., Brunswick (3)  
 Weymouth, Currier C., Eastmont, Farmington (4)  
 Weymouth, Raymond E., 194 Main St., Bar Harbor (5)  
 White, Henry O., 22 White St., Rockland (7)  
 White, Leland M., So. Main St., Caribou (2)  
 White, William J., 1 Mitchell Rd., South Portland (3)  
 Whitney, Byron V., 280 State St., Bangor (10)  
 Whitney, Ray L., Cape Porpoise (15)  
 Whittier, Alice A. S., 143 Neal St., Portland (3)  
 Whitworth, John E., 116 Hammond St., Bangor (10)  
 Wight, Donald G., 30 Mitchell Rd., South Portland (3)  
 Wilbur, Herbert T., Jr., P. O. Box 175, Southwest Harbor (5)  
 Willard, Harold N., Thayer Hospital, Waterville (6)  
 Williams, Edward P., 72 Main St., Houlton (2)

Williams, James A., 40 Pleasant St., Mechanic Falls (1)  
 Williams, Thomas W., 50 Union St., Ellsworth (5)  
 Williamson, Elizabeth E., Blue Hill (5)  
 Williamson, Russell G., Blue Hill, Mem. Hosp., Blue Hill (5)  
 Wilson, G. Ivan, 40 Court St., Houlton (2)  
 Wilson, Harry M., 944 Middle St., Bath (8)  
 Wilson, Robert D., Arthur R. Gould Mem. Hosp., Presque  
 Isle (2)  
 Wilson, Robert W., Veterans Administration, Togus (6)  
 Winchenbach, Francis A., 910 Washington St., Bath (8)  
 Wolfahrt, Eugene P., 338 Main St., Saco (15)  
 Wood, George W., III, 156 North Main St., Brewer (10)  
 Woodcock, Allan, 35 Second St., Bangor (10)  
 Woodcock, John A., 35 Second St., Bangor (10)  
 Woodman, Arthur B., 15 Johnson Rd., Falmouth Foreside (3)

Worthing, Verla E., Box A, Thomaston (7)  
 Wyman, David S., 47 Deering St., Portland (3)

## Y

Young, E. Stanley, Poland Spring (1)  
 Young, John, Bethel (9)

## Z

Zanca, Ralph, 86 Pine St., Lewiston (1)  
 Zeller, Alan W., 35 Main St., Damariscotta (8)  
 Zikel, Herbert M., High St., Wilton (4)  
 Zolov, Benjamin, 296 Congress St., Portland (3)



# Woman's Auxiliary to the Maine Medical Association

## ANDROSCOGGIN COUNTY

Andrews, Mrs. S. L. 35 White St., Lewiston  
 Archambault, Mrs. Philip L. 373 College St., Lewiston  
 Beaudet, Mrs. Simon C. 25 Webster St., Lewiston  
 Becaker, Mrs. Vincent H. 85 Wood St., Lewiston  
 Beegel, Mrs. Paul M. 80 Goff St., Auburn  
 Beliveau, Mrs. Bertrand A. 56 Howe St., Lewiston  
 Beliveau, Mrs. Romeo A. 89 Pine St., Lewiston  
 Bernard, Mrs. Romeo A. 26 Beacon St., Lewiston  
 Branch, Mrs. Charles F. 69 Gamage Ave., Auburn  
 Carrier, Mrs. John W. 53 Campus Ave., Lewiston  
 Chapin, Mrs. Milan A. 237 Turner St., Auburn  
 Chevalier, Mrs. Paul R. 353 Pine St., Lewiston  
 Clapp, Mrs. Waldo A. 215 College St., Lewiston  
 Clapperton, Mrs. Gilbert 21 Ryder St., Lewiston  
 Cloutier, Mrs. Wilfrid A. 210 Sabattus St., Lewiston  
 Cox, Mrs. William V. 82 Gamage Ave., Auburn  
 DeCosta, Mrs. Donald A. Poland  
 Dycio, Mrs. George 55 Broad St., Auburn  
 Fahey, Mrs. William J. 17 Frye St., Lewiston  
 Fishman, Mrs. Louis N. 223 Lake St., Auburn  
 Flanders, Mrs. Merton N. 370 Main St., Lewiston  
 Fortier, Mrs. Paul J. B. Barron Ave., Lewiston  
 Friend, Mrs. John W. 70 Western Ave., Auburn  
 Frost, Mrs. Robert A. 108 Summer St., Auburn  
 Gauvreau, Mrs. Horace L. 69 Horton St., Lewiston  
 Gauvreau, Mrs. Norman 69 Fair St., Lewiston  
 Giguere, Mrs. Eustache N. 98 Webster St., Lewiston  
 Goldman, Mrs. Morris E. 524 Main St., Lewiston  
 Goodwin, Mrs. Ralph A., Sr. 56 Denison St., Auburn  
 Goodwin, Mrs. Ralph A., Jr. 48 Grandview Ave., Auburn  
 Green, Mrs. Ross W. R.F.D. No. 2, Auburn  
 Greene, Mrs. John P. R.F.D. No. 2, Auburn  
 Greene, Mrs. Merrill S. F. 466 Main St., Lewiston  
 Gross, Mrs. Leroy C. 19 Goff St., Auburn  
 Haas, Mrs. Rudolph 484 Main St., Lewiston  
 Hannigan, Mrs. Charles A. 85 Goff St., Auburn  
 Hiebert, Mrs. Joelle C., Jr. Box 148, Norway  
 Higgins, Mrs. Everett C. 149 College St., Lewiston  
 Hirshler, Mrs. Max 25 Bardwell St., Lewiston  
 Horsman, Mrs. Donald H. 50 Goff St., Auburn  
 James, Mrs. Chakmakis 47 Howe St., Lewiston  
 James, Mrs. John A. R.F.D. No. 2, Auburn  
 Konecki, Mrs. John T. R.F.D. No. 2, Auburn  
 LaFlamme, Mrs. Paul J. 106 Russell St., Lewiston  
 Lemaitre, Mrs. Paul G. 268 Webster St., Lewiston  
 Lichter, Mrs. Horace A. Maple Hill, Auburn  
 Lidstone, Mrs. Frederick B. R.F.D. No. 2, Auburn  
 Martel, Mrs. Cyprien L., Jr. 24 Frye St., Lewiston  
 Mendes, Mrs. Joseph M. 221 Pleasant St., Lisbon Falls  
 Methot, Mrs. Frank P. 1 Bellegarde Circle, Lewiston  
 Milazzo, Mrs. John 42 Elm St., Auburn  
 Miller, Mrs. Clark F. 46 Madison St., Auburn  
 Morissette, Mrs. Russell A. 69 Western Promenade, Auburn  
 Nadeau, Mrs. J. Paul 91 Pine St., Lewiston  
 O'Connell, Mrs. George B. 79 Shepley St., Auburn  
 Potts, Mrs. Ronald S. 18 Ware St., Lewiston  
 Proulx, Mrs. Harvey J. 435 East Ave., Lewiston  
 Rand, Mrs. Carleton H. 166 College St., Lewiston  
 Rando, Mrs. Joseph J. 186 Davis Ave., Auburn  
 Rock, Mrs. Daniel A. 477 Main St., Lewiston  
 Shems, Mrs. Albert 487 Main St., Lewiston  
 Shields, Mrs. Daniel R. R.F.D. No. 2, Auburn  
 Spear, Mrs. William 107 Main St., Lisbon Falls  
 Steele, Mrs. Charles W. 1 Wakefield St., Lewiston  
 Sweatt, Mrs. L. A. 48 Drummond St., Auburn  
 Swett, Mrs. Alfred E. 308 Minot Ave., Auburn

Tchao, Mrs. Jou S. West Auburn  
 Thacher, Mrs. Henry C. Upper Turner St., Auburn  
 Tibbetts, Mrs. Otis B. R.F.D. No. 2, Auburn  
 Twaddle, Mrs. Gard W. 57 Goff St., Auburn  
 Wakefield, Mrs. Robert D. R.F.D. No. 2, Auburn  
 Webber, Mrs. Wedgwood P. 376 Main St., Lewiston  
 Young, Mrs. E. Stanley P.O. Box 256, Gray

## AROOSTOOK COUNTY

Aungst, Mrs. Melvin R. 4 High St., Fort Kent  
 Brown, Mrs. Stephen S. Mars Hill  
 Collins, Mrs. H. Douglas Home Farm Rd., Caribou  
 Donahue, Mrs. Clement L. 13 Collins St., Caribou  
 Donahue, Mrs. Gerald H. 52 Dudley St., Presque Isle  
 Etscovitz, Mrs. Eli A. Home Farm Rd., Caribou  
 Gormley, Mrs. Eugene G. 46 High St., Houlton  
 Gregory, Mrs. Frederick J., Sr. Caribou  
 Hayward, Mrs. I. Mead Caribou  
 Johnson, Mrs. R. Paul Hall St., Fort Kent  
 Kimball, Mrs. Herrick C. Presque Isle Rd., Fort Fairfield  
 Pendleton, Mrs. Arthur D. 7 Forest Ave., Fort Fairfield  
 Pines, Mrs. Philip 22 Long Rd., Limestone  
 Rideout, Mrs. Samuel 2 Depot St., Fort Fairfield  
 Sterlin, Mrs. Andre High St., Fort Kent  
 Toussaint, Mrs. Leonid G. 13 Pleasant St., Fort Kent

## CUMBERLAND COUNTY

Agan, Mrs. Robert W. 3 Rocky Hill Rd., Cape Elizabeth  
 Allen, Mrs. Donald E. Sebago Lake  
 Ansell, Mrs. Harvey B. 136 Baxter Blvd., Portland  
 Aranson, Mrs. Albert 177 Caleb St., Portland  
 Asherman, Mrs. Edward G. 275 Falmouth Rd., Falmouth  
 Babalian, Mrs. Leon Surf Rd., Cape Cottage  
 Bacastow, Mrs. Merle S. 21 Ivie Rd., Cape Elizabeth  
 Baldini, Mrs. Elio 89 West St., Portland  
 Baldwin, Mrs. Warren C. 24 Andrews Rd., Falmouth Foreside  
 Bennet, Mrs. Eben T. Shore Rd., Cape Elizabeth  
 Bidwell, Mrs. Robinson L. 24 Casco Ter., Falmouth Foreside  
 Bischoffberger, Mrs. John M. Naples  
 Blaisdell, Mrs. Elton R. 35 Penrith Rd., Portland  
 Blumberg, Mrs. Edward Box C, Pownal  
 Bonney, Mrs. James H. 229 Vaughan St., Portland  
 Bove, Mrs. Louis G. 95 West St., Portland  
 Bowman, Mrs. Peter W. Box C, Pownal  
 Branson, Mrs. Sidney R. Gray Rd., South Windham  
 Broggi, Mrs. Frank S. 18 Neal St., Portland  
 Brown, Mrs. Douglas H. Birchwood Rd., Cape Elizabeth  
 Brown, Mrs. Luther A. 13 Deering St., Portland  
 Burnett, Mrs. Claude A., Jr. Delano Park, Cape Elizabeth  
 Burrage, Mrs. William C. 53 Chadwick St., Portland  
 Capron, Mrs. Charles W. Hunnewell Rd., Scarborough  
 Chase, Mrs. George O. 67 College St., Portland  
 Christensen, Mrs. Harry E. South Freeport  
 Clark, Mrs. Frederick B. 112 Foreside Rd., Falmouth  
 Clarkin, Mrs. Charles P. 64 Brookside Rd., Portland  
 Cole, Mrs. Donald P. 55 Cottage Farms Rd., Cape Elizabeth  
 Crane, Mrs. Lawrence Dean's Way, Cumberland Foreside  
 Cummings, Mrs. George O. Sr. 583 Shore Rd., Cape Elizabeth  
 Cummings, Mrs. George O., Jr. 13 West St., Portland  
 Curtis, Mrs. Harry L. 45 Wellington Rd., Portland  
 D'Andrea, Mrs. Anthony L. Fall Lane, Portland  
 Darche, Mrs. Albert A. 143 King St., Westbrook  
 Davies, Mrs. Lloyd G. 78 Main St., Fryeburg  
 Davis, Mrs. Harry E. Bramhall Field, Falmouth Foreside  
 Derry, Mrs. G. Hermann 888 Shore Rd., Cape Elizabeth

Dionne, Mrs. Maurice J. Pleasant Hill Rd., Brunswick  
Doby, Mrs. Tibor 16 Wood Rd., Cape Elizabeth  
Dooley, Mrs. Francis M. 53 Deering St., Portland  
Dorsey, Mrs. F. Donald 82 Foreside Rd., Falmouth  
Doughinett, Mrs. Otis J. Maple Ave., Scarborough  
Drake, Mrs. Emerson H. State Rd., Cumberland Foreside  
Drake, Mrs. Eugene H. County Rd., South Gorham  
Drexler, Mrs. James E. Ward Town Rd., Freeport  
Drummond, Mrs. Joseph B. Ship Channel Rd., So. Portland  
Dunham, Mrs. Carl E. 1122 Washington Ave., Portland  
Dyhrberg, Mrs. Norman E. 331 Main St., Cumberland Mills  
Earnhardt, Mrs. Joseph B. 55 Stroudwater St., Westbrook  
Eppinger, Mrs. Ernst 52 Belmont St., Portland  
Fagone, Mrs. Francis A. 173 Bolten St., Portland  
Ferguson, Mrs. Franklin F. Surf Rd., Cape Cottage  
Fish, Mrs. Nicholas State Rd., Cumberland Foreside  
Fogg, Mrs. Philip S., Jr. 173 Pleasant Ave., Portland  
Fox, Mrs. Francis H. 83 West St., Portland  
Galen, Mrs. Robert S. 22 MacMillan Dr., Brunswick  
Gates, Mrs. Clifford W. Flaggy Meadow Rd., Gorham  
Geer, Mrs. Charles R. 212 Vaughan St., Portland  
Geer, Mrs. George L., Jr. 58 Clifford St., South Portland  
Gehring, Mrs. Edwin W. 284 Ocean Ave., Portland  
Gibbons, Mrs. John F. Maiden Cove Lane, Cape Elizabeth  
Glassmire, Mrs. Charles R. 20 Drew Rd., South Portland  
Goduti, Mrs. Richard J. 16 Brown St., Portland  
Good, Mrs. Philip G. 126 Fickett St., South Portland  
Hallett, Mrs. George W., Jr. Shore Rd., Cape Elizabeth  
Haney, Mrs. Oramel 74 Deering St., Portland  
Hanley, Mrs. Daniel F. 58 Federal St., Brunswick  
Hawkes, Mrs. Richard S. 174 Longfellow St., Portland  
Hecht, Mrs. Henry 326 Stevens Ave., Portland  
Heifetz, Mrs. Ralph 112 Chenery St., Portland  
Hill, Mrs. Douglas R. 2 State Ave., Cape Elizabeth  
Hinckley, Mrs. Harris Shore Rd., Cape Elizabeth  
Holt, Mrs. C. Lawrence 230 Foreside Rd., Falmouth Foreside  
Hudson, Mrs. Henry A. 11 Gage St., Bridgton  
Ives, Mrs. Howard R. 56 Bowdoin St., Portland  
Jacobson, Mrs. Payson B. 295 Brighton Ave., Portland  
Johnson, Mrs. Albert C. Shore Acres, Cape Elizabeth  
Johnson, Mrs. Henry P. 16 Sheffield St., Portland  
Johnson, Mrs. Oscar R. 18 Deering St., Portland  
Kent, Mrs. Stanley W. 15 Payson Rd., Falmouth Foreside  
Knowles, Mrs. Robert M. Cumberland Foreside  
Lape, Mrs. C. Philip 132 Chadwick St., Portland  
Leary, Mrs. Gerald C. 7 Redlon Rd., Portland  
Lincoln, Mrs. John R. 120 Woodville Rd., Falmouth  
Logan, Mrs. G. E. C. 44 Shore Line Dr., Falmouth  
Lorimer, Mrs. Robert V. 1 Ocean Rd., South Portland  
Lovely, Mrs. David K. 52 Berkeley St., Portland  
Mack, Mrs. Francis X. 1473 Westbrook St., Portland  
MacVane, Mrs. William L., Jr. 25 Storer St., Portland  
Maltby, Mrs. George L. Bramhall Field, Falmouth Foreside  
Manol, Mrs. Jack 157 Pine St., Portland  
Marshall, Mrs. Donald F. Surf Rd., Cape Cottage  
Marshall, Mrs. Richard A. 3 Parsons Rd., Portland  
Marston, Mrs. Paul C. Kezar Falls  
Martin, Mrs. Thomas A. 1415 Forest Ave., Portland  
Matthews, Mrs. Edward C. 127 Neal St., Portland  
Mautner, Mrs. Hans V. 44 Lafayette St., Yarmouth  
Mazzone, Mrs. Giovanni 15 Fall Lane, Portland  
McCann, Mrs. Eugene C. 110 Foreside Rd., Falmouth  
McCrum, Mrs. Philip H. 15 Fairlawn Ave., South Portland  
McIntire, Mrs. Barron F., Jr. 13 W. Elm St., Yarmouth  
McLean, Mrs. E. Allan 331 Foreside Rd., Falmouth Foreside  
McManamy, Mrs. Eugene P. Surf Rd., Cape Cottage  
McMichael, Mrs. Morton Pope Rd., Windham Hill  
Mohlar, Mrs. Robert G. 5 Atwood Lane, Brunswick  
Monaghan, Mrs. Stephen E. 65 Drew Rd., South Portland  
Monkhouse, Mrs. William A. 29 Bowdoin St., Portland  
Morrison, Mrs. Alvin A. 165 Glenwood Ave., Portland  
Moulton, Mrs. Albert W., Jr. 97 Vaughan St., Portland  
Nichols, Mrs. Estes 59 West St., Portland  
O'Donnell, Mrs. Eugene E. 12 Cottage Farms Rd., Cape Elizabeth  
Olmsted, Mrs. Burton L. 8 Rock Wall Lane, Cape Elizabeth  
Orberton, Mrs. Everett A. 45 Channel Rd., South Portland  
Osber, Mrs. Harold L. 66 Chadwick St., Portland  
Ottum, Mrs. Alvin E. 77 Falmouth Rd., Falmouth  
Pawle, Mrs. Robert H. 8 Walcott Ave., Falmouth  
Penta, Mrs. Walter E. 316 Woodford St., Portland  
Perkins, Mrs. Niles L., Jr. 135 Clinton St., Portland  
Pettersen, Mrs. Herman C. Chebeague Island  
Poliner, Mrs. Irving J. Hillcrest Rd., Cape Elizabeth  
Polisner, Mrs. Saul R. 143 Vaughan St., Portland  
Porter, Mrs. Joseph E. 53 Falmouth Rd., Falmouth  
Proudian, Mrs. Paul O. 88 Forest St., Westbrook  
Pudor, Mrs. G. A. 15 Sheffield St., Portland  
Ray, Mrs. Ferris S. 23 Brown St., Falmouth Foreside  
Robinson, Mrs. Hugh P. Waites Landing, Falmouth Fortside  
Sager, Mrs. George F. Shore Rd., Cape Elizabeth  
Santoro, Mrs. Domenico A. 43 Deering St., Portland  
Sapiro, Mrs. Howard M. 44 Pitt St., Portland  
Sawyer, Mrs. Howard P., Jr. 672 Ocean Ave., Portland  
Selvage, Mrs. Irving L., Jr. 88 Ivie Rd., Cape Elizabeth  
Shapiro, Mrs. Morrill 95 Caleb St., Portland  
Skillin, Mrs. Charles E. Sea Cove Rd., Cumberland Foreside  
Stebbins, Mrs. Arthur P. 996 Sawyer St., So. Portland  
Stephenson, Mrs. Richard B. 12 Woodland Rd., Cape Elizabeth  
Stevens, Mrs. Theodore M. 9 Ricker Park, Portland  
Storer, Mrs. Daniel P. 108 Fessenden St., Portland  
Sylvester, Mrs. Allan W. 396 Ocean Ave., Portland  
Sylvester, Mrs. Stanley B. 1346 Westbrook St., Portland  
Tabachnick, Mrs. Henry M. 110 Park Ave., Portland  
Taylor, Mrs. William F. Providence Ave., Falmouth Foreside  
Telfeian, Mrs. Alphonse 5 Brookside Rd., Portland  
Tetreau, Mrs. William J. 25 Fall Lane, Portland  
Thaxter, Mrs. Langdon T. State Rd., Cumberland Foreside  
Thompson, Mrs. Philip P., Jr. 7 Ship Channel Rd., So. Portland  
Titherington, Mrs. John B. 97 Brook Rd., Falmouth  
Turcotte, Mrs. Guy N. 6 Oakwood Rd., Cape Elizabeth  
Turgeon, Mrs. Raphael F. 68 Lyman St., Westbrook  
Urjanis, Mrs. Janis Box C, Pownal  
Van Lonkhuyzen, Mrs. Maurice 1009 Shore Rd., Cape Elizabeth  
Webber, Mrs. Isaac M. Penrith Rd., Portland  
Wellington, Mrs. J. Foster 396 Brighton Ave., Portland  
White, Mrs. William J. 1 Mitchell Rd., South Portland  
Wight, Mrs. Donald G. 30 Mitchell Rd., South Portland  
Woodman, Mrs. George M. 826 Main St., Westbrook  
Wyman, Mrs. David S. 7 Bayview Ave., South Portland  
Zolov, Mrs. Benjamin 430 Baxter Blvd., Portland

### FRANKLIN COUNTY

Bowne, Mrs. Hays G. 9A Main St., Farmington  
Brinkman, Mrs. Harry 47 Perham St., Farmington  
Chase, Mrs. Philip B. 36 Main St., Farmington  
Colley, Mrs. Maynard B. Main St., Farmington  
Covert, Mrs. S. Burton Kingfield  
Duffy, Mrs. Wallace H. 100 Main St., Farmington  
Eastman, Mrs. Charles W. 15 Millet St., Livermore Falls  
Fiorica, Mrs. Gaetano T. 12 Church St., Chisholm  
Floyd, Mrs. Paul E. 2 Middle St., Farmington  
Marsters, Mrs. David W. Phillips  
Pratt, Mrs. Harold S. 18 Church St., Livermore Falls  
Reed, Mrs. James W. 18 Main St., Farmington  
Rowe, Mrs. Gunther H. 42 Main St., Livermore Falls  
Thompson, Mrs. Cecil F. Phillips

### KENNEBEC COUNTY

Ashley, Mrs. T. A. Sylvan Rd., Farmingdale  
Barnard, Mrs. John M. H. Malta Lane, Augusta  
Barron, Mrs. Richard E. East Monmouth  
Canal, Mrs. Ory D. Augusta State Hospital, Augusta  
Castellanos, Mrs. Jose Augusta State Hospital, Augusta  
Crawford, Mrs. Albert S. Veterans Adm., Togus  
Crawford, Mrs. Joseph R. 86 Winthrop St., Augusta  
Dachslager, Mrs. Philip 55 Capitol St., Augusta  
Daniels, Mrs. Donald H. Mill Pond, Readfield  
Darlington, Mrs. Brinton T. Westwood Rd., Augusta  
Denison, Mrs. John D. 105 Brunswick Ave., Gardiner  
Dunn, Mrs. Robert H. 105 Dresden Ave., Gardiner  
Fallon, Mrs. Louis F. 52 Lincoln Ave., Augusta  
Farrell, Mrs. Chalmers G. 68 Central St., Gardiner  
Fisher, Mrs. Samson 173 Main St., Waterville

Giddings, Mrs. Lane 76 Purinton Ave., Augusta  
 Gingras, Mrs. Adolphe J. 113 Northern Ave., Augusta  
 Gingras, Mrs. Napoleon J. 124 State St., Augusta  
 Gould, Mrs. George I. Richmond  
 Jackson, Mrs. Elmer H. 47 Chapel St., Augusta  
 Landwehr, Mrs. George R. 111 Water St., Augusta  
 Lepore, Mrs. Anthony E. 76 School St., Gardiner  
 Mathews, Mrs. Hugh J., Jr. 75 Brunswick Ave., Gardiner  
 McLaughlin, Mrs. Clarence R. 152 Brunswick Ave., Gardiner  
 McLaughlin, Mrs. Ivan E. Lewiston Rd., Gardiner  
 McQuillan, Mrs. Arthur H. Pond Road, Oakland  
 McWethy, Mrs. Wilson H. 22 Sewall St., Augusta  
 Melendy, Mrs. Oakley A. Westwood Rd., Augusta  
 Milliken, Mrs. Howard H. 105 Second St., Hallowell  
 Monsiva's, Mrs. Alfred Augusta State Hospital, Augusta  
 Moore, Mrs. Arnold W. 112 Eastern Ave., Augusta  
 Morris, Mrs. Craig W. Church Hill Rd., Augusta  
 O'Connor, Mrs. Francis J. 4 Woodlawn St., Augusta  
 Ohler, Mrs. Robert L. East Winthrop  
 Pratt, Mrs. Loring W. 37 Lawrence Ave., Fairfield  
 Richards, Mrs. Lee W., Jr. 89 Winthrop St., Augusta  
 Robertson, Mrs. George J. 33 College Ave., Waterville  
 Sanders, Mrs. Stephen W. 120 Main St., Winthrop  
 Saunders, Mrs. Allen I. Ferry Rd., R.F.D. 2, Augusta  
 Schumacher, Mrs. William E. 14 Westwood Rd., Augusta  
 Shippee, Mrs. James N. Main St., Winthrop  
 Sleeper, Mrs. Francis H. Box 724, Augusta State Hospital, Augusta  
 Sommerfeld, Mrs. Kurt A. 182 Dresden Ave., Gardiner  
 Stinchfield, Mrs. Allan J. 6 Warren St., Hallowell  
 Weltman, Mrs. Joseph S. Veterans Administration, Togus

### KNOX COUNTY

Adams, Mrs. Lester 9 Knox St., Thomaston  
 Dennison, Mrs. Frederick C. 52 Main St., Thomaston  
 Eddy, Mrs. Robert H. 130 Chestnut St., Camden  
 Heath, Mrs. Parker, Jr. 58 Washington St., Camden  
 Hochschild, Mrs. Hugo 33 Main St., Thomaston  
 Hopping, Mrs. John S. Union  
 Hunter, Mrs. Albert L. Sherman's Pt., Camden  
 Jameson, Mrs. C. Harold Chestnut St., Camden  
 Kibbe, Mrs. Frank W. Lincolnville  
 King, Mrs. Merrill J., Sr. 89 Talbot Ave., Rockland  
 Lawry, Mrs. Oram R., Jr. 96 Limerock St., Rockland  
 McLellan, Mrs. William A. 87 Chestnut St., Camden  
 Millington, Mrs. Paul A. 44 Mountain St., Camden  
 Morse, Mrs. Edward K. 39 High St., Camden  
 Root, Mrs. John A. 111 Beech St., Rockland  
 Soule, Mrs. Gilmore W. 616 Old County Rd., Rockland  
 Tounge, Mrs. Harry G., Jr. 12 Union St., Camden  
 Ward, Mrs. William W. 76 Limerock St., Rockland  
 Wasgatt, Mrs. Wesley N. 41 Talbot Ave., Rockland  
 White, Mrs. Henry O. Harden Ave., Camden

### LINCOLN-SAGADAHOC COUNTY

Akar, Mrs. Hamdi 17 Grove St., Bath  
 Belknap, Mrs. Samuel L. Damariscotta  
 Bostwick, Mrs. George W. Newcastle  
 Gregory, Mrs. Philip O. Boothbay Harbor  
 Hutchins, Mrs. Deane L. 69 Townsend Ave., Boothbay Harbor  
 Kershner, Mrs. Warren E. 57 Green St., Bath  
 Kinder, Mrs. Edward L., Jr. 1027 Washington St., Bath  
 Mullin, Mrs. Ann 3 Allen's Lane, Bath  
 Powell, Mrs. Ralph C. Damariscotta  
 Proctor, Mrs. Thomas E. Boothbay Harbor  
 Smith, Mrs. Jacob 709 High St., Bath  
 Smith, Mrs. Joseph I. 940 Washington St., Bath  
 Stott, Mrs. Ardenne A. 37 Park St., Bath  
 Winchenbach, Mrs. Francis A. 910 Washington St., Bath

### OXFORD COUNTY

Akerberg, Mrs. Ake Maple St., South Paris  
 Aucoin, Mrs. Peter B. 25 Rumford Ave., Rumford  
 Bean, Mrs. H. Richard Crocket Ridge, Norway  
 Defoe, Mrs. Garfield G. Dixfield

Dixon, Mrs. Walter G. 16 Deering St., Norway  
 Elsmore, Mrs. Dexter E. Dixfield  
 Greene, Mrs. John A. 90 Congress St., Rumford  
 Harper, Mrs. Harry L. 17 Main St., South Paris  
 Howard, Mrs. Henry M. 105 Franklin St., Rumford  
 Hubbard, Mrs. Roswell E. Waterford  
 MacDougall, Mrs. James A. 303 Penobscot St., Rumford  
 McCormack, Mrs. Roland L. 12 Bridge St., Norway  
 Nangle, Mrs. Thomas P. West Paris  
 Nelson, Mrs. Chesley W. 121 Main St., Norway  
 Rowe, Mrs. Linwood M. 246 Penobscot St., Rumford

### PENOBSCOT COUNTY

Adams, Mrs. Asa C. 99 Forest Ave., Orono  
 Adams, Mrs. Winford C. R.F.D. 5, Brewer  
 Ames, Mrs. Forrest B. 13 Long Meadow Dr., Brewer  
 Babcock, Mrs. Edward B. 46 Norway Rd., Bangor  
 Barrett, Mrs. Robert J., Jr. 371 Union St., Bangor  
 Blackburn, Mrs. Nelson P. Winterport  
 Blaisdell, Mrs. William B. Penobscot Ter., Brewer  
 Brown, Mrs. Lloyd 427 Garland St., Bangor  
 Burke, Mrs. Paul W. 5 High St., Newport  
 Butler, Mrs. Harry 77 Broadway, Bangor  
 Clement, Mrs. James D., Jr. 158 Kenduskeag Ave., Bangor  
 Clough, Mrs. Dexter J., 2nd River Rd., Bucksport  
 Comeau, Mrs. Wilfred 115 Main St., Orono  
 Cornell, Mrs. Robert C. 118 Forest Ave., Orono  
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 31 Summer St., Kennebunk  
 5 Edmond St., Springvale  
 35 Summer St., Kennebunk  
 Bar Mills  
 200 Granite St., Biddeford  
 415 Main St., Saco  
 319 Main St., Saco  
 York  
 24 Portland Ave., Old Orchard  
 34 Union St., Biddeford  
 13 Bacon St., Biddeford  
 North Berwick  
 York  
 23 Weymouth St., Saco  
 99 Main St., Kennebunk  
 7 Redlon Rd., Portland  
 410 Main St., Sanford  
 York  
 338 Main St., Saco  
 4 Rolles St., Springvale  
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 38 May St., Biddeford  
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